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CONGENITAL ANTEVERSION OF THE NECK OF THE FEMUR

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CONGENITAL anteversion of the neck of the femur without dislocation of the hip is a relatively common congenital deformity. This condition is scarcely mentioned in medical literature and no record of it is found in the standing textbooks on orthopedic surgery. In most cases, the deformity is of insufficient degree to produce symptoms of any consequence. Rarely the congenital anteversion of the femoral necks is of such a degree that it does result in deformities of gait. It is for this reason that it was deemed advisable to describe certain clinical aspects of this condition.

The easiest way to understand what is meant by "anteversion of a femoral neck" is to examine a femur carefully. If a femur is placed with the posterior aspect downwards on a flat surface so that the medial and lateral femoral condyles and the posterior aspect of the greater trochanter rest upon this surface, it is noted that a pin drilled through the center of the femoral head and neck makes an angle with the plane of this surface of 0° to 15° in the normal adult femur (Figure 1). In the condition known as "congenital anteversion of the femoral neck," the angle between the head and neck of the femur and the flat surface noted above upon which the femur rests has an angulation in excess of the 15° mentioned. In fact, this angulation may, in certain instances, approach or reach 60°.

Medial and lateral rotation movements of the normal hip are limited by the relationship of the head and neck of the acetabulum. Medial rotation of the hip is that movement in which the anterior surface of the thigh is rotated toward the midline, while lateral rotation is that movement in which the anterior surface of the thigh is rotated away from the midline. The anterior surface of the femoral neck will come in contact with the anterior surface of the acetabulum with medial rotation movements and the posterior surface of the neck will come in contact with the posterior surface of the acetabulum with lateral rotation, thus limiting such movements. It is obvious that in a case of congenital anteversion of the femur, the hip will have excessive medial rotation and diminished or absent lateral rotation.

An individual cannot walk in a normal manner without lateral rotation movements of the hips, because, in the normal walking gait, lateral rotation of the hips occurs simultaneously with advancing the foot and leg forward on the same side. If a lateral rotation motion of the hip is not possible, advancing the pelvis forward on that side results in an apparent medial rotation of this hip, giving a peculiar gait, or else the individual must walk in a very

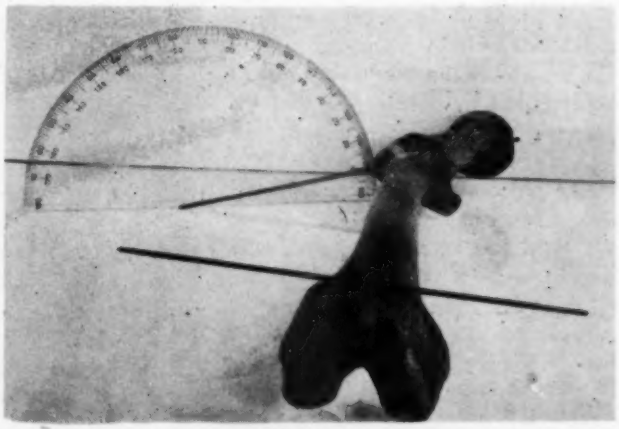

Figure 1

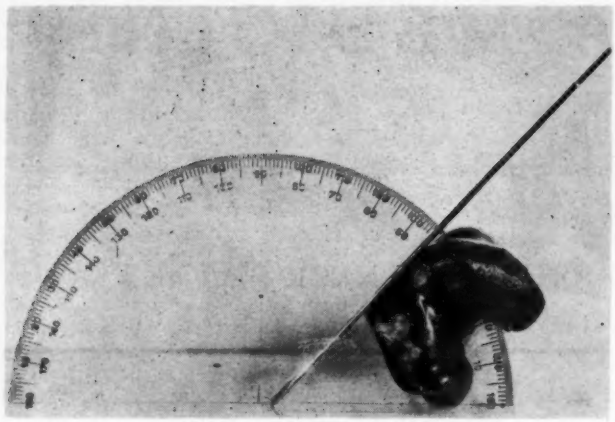

Figure 2
55° Lateral Rotation of Right Hip

stiff manner, keeping the pelvis at right angles to the direction of movement.

SYMPTOMS

The patient with congenital anteversion of the femoral necks (since the condition in our experience is bilateral) is brought to the doctor with the complaint that the child is knock-kneed, that the "feet turn in," or that the patient has a peculiar gait. The parents state that the deformity was noted soon after the child began to walk. The parents may state that the child has difficulty in sitting "tailor fashion" or that the child sits on the inside of his heels with the outside of his feet flat against the floor. In some cases the parents complain that the patient is unable to cross the knees in an ordinary manner. It is not unusual for the parents to describe having taken the child to several doctors who have attempted to correct the deformity by wedging the heels of the shoes or by applying braces to the legs or feet without benefit. In our experience the parents are almost always unaware of the fact that the deformity is in the hip region.

PHYSICAL EXAMINATION

The physical examination ordinarily is entirely negative except that the child has excessive medial rotation movements of the hip and absent or decreased lateral rotation movements. In order to determine this fact accurately the child should be stripped and placed supine on a firm flat table. The pelvis should be kept horizontal with the table and not permitted to turn, thus giving a false impression of rotary movements. The examiner should grasp the legs or feet and medially rotate both thighs, noting the degree of angulation between an imaginary plane parallel with the anterior surface of the patella (since this plane is essentially parallel with the plane noted previously with the femora lying on a flat surface) and the plane of the table (Figures 2 and 3). For example, if medial rotation may be carried out to such a degree that the knee caps directly face each other, each may be said to have approximately 90° of medial rotation. Lateral rotation is determined in the same manner by rotating the thighs the maximum amount laterally and noting the degree of angulation in the same manner as that of medial rotation. For example, if lateral rotation may be carried out to such a degree that the knee caps are on exactly opposite sides of the femora, then lateral rotation would be approximately 90°. If lateral rotation may be carried out so that the knee caps will face only directly forward (or upwards in this case), the degree of lateral rotation of the hips is approximately zero. If the degree of medial rotation greatly exceeds that of lateral rotation, the examiner may feel assured that some anteversion of the femoral neck is present unless there is some other condition to explain this abnormality, such as a fractured femur which has healed in mal-union.

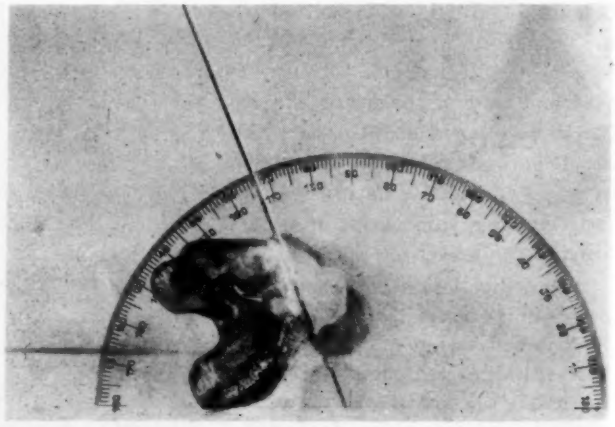

Figure 3
67° of Medial Rotation of the Right Hip

It is important not to gauge the degree of rotation of the hips by the position of the tibial tuberosities, because a certain amount of rotary movement may take place in the knee joints and it is even more important not to gauge the degree of rotation of the hips by the position of the feet since considerable rotatory motion may take place in the subtalar and mid-tarsal joints. The patient should be observed walking while wearing no clothing so that the position of the thighs may be clearly seen. By this means, it may be determined that a decrease in lateral rotation of the hips is present which will give the patient a peculiar swinging type of gait.

In the average adult without obvious hip disease or some injury to the femora, the degree of lateral rotation of the hips exceeds the medial rotation. Our clinical observations, which are not yet complete, indicate that the same may not be true in children, and that more variation is present with them. This is confirmed by the studies of Kingsley & Olmstead.

X-RAY EXAMINATION

Cases suspected of having congenital anteversion of the femoral necks should have anterior-posterior radiographs of both hips taken with the hips in maximum medial rotation, maximum lateral rotation and in a neutral position with the hips in no rotation. Most radiologists are not familiar with this condition and will report the hips as being normal. In anterior-posterior radiographs of normal adult hips, the femoral necks appear to have the greatest length when they are parallel to the film which means when they are in 0° to 15° of medial rotation. In the condition under discussion, the femoral necks will have their greatest length when the hips are held in considerable medial rotation, and they will have the least length when the hips are held in maximum lateral rotation.

CLINICAL COURSE

Whether or not this condition would disappear spontaneously with the growth of the child has not been fully determined, but in the case report to follow, no significant improvement was noted after observing the child for twenty-three months. Conservative treatment by wearing "twisters" has been tried by the author, but the follow-up period is inadequate. Treatment of the condition by use of heel wedges and other appliances apparently is inadequate. The following case was treated surgically with very satisfactory results.

REPORT OF CASE

This 3½ year old white girl was first seen June 6, 1947, because her parents complained that she was knock-kneed. The deformity had been noted since the child started to walk at ten months of age. She had a wobbly gait at that time and seemed to be "pigeon-toed." The deformity appeared to increase with age. She was taken to doctors who prescribed heel wedges, but these failed to alleviate the condition. The mother noted that the child often assumed a peculiar position, sitting on the medial surfaces of the ankles with the lateral surfaces against the floor. She did not complain of pain.

There was no family history of hip disease of any kind. The patient weighed five pounds at birth, and delivery was said to have been normal and at term. The child was given oleum percomorphum regularly. She had chicken-pox when two years of age and no other childhood disease. She was able to hold her head up at four months, to sit up at five months, and to walk when about ten months of age.

EXAMINATION

The examination on June 6, 1947, revealed no abnormalities except for the lower extremities. There was no clinical evidence of dislocation of the hips. The lower extremities were symmetrical and equal in length and circumference. The hips had an entirely normal range of motion except for rotation. Medial rotation exceeded 90°, bilaterally, but lateral rotation was only 25° to 30°, bilaterally. Her lower-extremities were straight, there being neither knock-knee nor bow-leg deformities. There was no unusual relaxation of the knee joints. Her feet appeared perfectly normal in the lying or sitting positions. There was no tibial torsion deformity. When the patient stood, she appeared to be knock-kneed, but obviously this was due to the fact that she held her hips in a medially rotated position while standing. She walked with a peculiar swinging gait with the feet apparently adducted.

X-RAY EXAMINATION

Radiographs of both hips with the hips in a neutral position (Figure 4) revealed no abnormality except that the femoral necks seemed unusually short and a coxa valga deformity appeared to be present. A radiograph taken with the hips in maximum medial rotation (Figure 5) revealed that the femoral necks were much longer than they appeared to be in the neutral position and that coxa valga deformity was not present. The mother was informed of the nature of the condition. She was told that femoral osteotomies might prove necessary at some later

date if the condition did not correct itself spontaneously.

CLINICAL COURSE

The patient was re-examined September 12, 1947, and no difference in the range of motion was noted. Both hips had 90° or slightly more of medial rotation, while lateral rotation did not exceed 20°. On March 8, 1948, re-examination revealed no change in the patient's condition and on May 4, 1949, clinical photographs were taken (Figures 6 and 7). The peculiar gait was still present. She had a very slight genu-varum deromity. The range of passive motion of her hips was as follows: abduction 45°, adduction 45°, flexion 30°, extension 180°, medial rotation 90°, latetral rotation 5°. Her ankles dorsiflexed to 80. ° Her feet were flexible and no foot deformities were noted.

On May 12, 1949 surgical operation was carried out in the following manner. Steinman pins were inserted at the middle thirds of the femora and at the lateral and anterior surfaces so that

Figure 4

Figure 5

Figure 6
Patient standing in maximum hip lateral rotation.

Figure 7
Patient standing in maximum medial hip rotation.

these pins made an angle of approximately 55° with the horizontal plans while the patient was lying supine with the hips in neutral rotation. Then, subtrochanteric osteotomies were carried out and the distal femoral fragments were ro-

tated laterally so that the Steinman pins were horizontal. The patient was immobilized in this position in a double hip spica.

On July 18, 1949, both casts were removed, the osteotomies having healed firmly. The patient was kept in bed until she had regained good strength of her quadriceps muscles and then was permitted to walk at increased intervals.

The patient continued to improve although she did walk in a rather unsteady manner for several weeks. Her parents were concerned at first because her hips seemed to be wider than they were before surgery. Post-operative x-ray examination, November 14, 1949 (Figure 8) revealed solid union of the osteotomies with normal appearance of the hips in neutral rotation. At the last examination on January 26, 1950, the patient had improved greatly and the parents were pleased with the results. The patient walked in a normal manner, the previously noted limp having entirely disappeared. The gluteus medius muscles were normal. The range of motion of her hips, passively or actively, was as follows: flexion, right 30°, left 30°; extension, right 180°, left 180°; abduction, right 45°, left 45°; adduction, right 60°, left 50°; medial rotation, right 50°, left 45°; lateral rotation, right 45°, left 50°. The operative scars showed a tendency to be keloidal. When the patient stood with her ankles touching each other, her knees were separated by an amount not exceeding 1/16". Clinical photographs were taken on this date (Figures 9 and 10).

Figure 8

SUMMARY

Clinical aspects of congenital anteversion of the femora neck are discussed and one case report is given, indicating that the condition may produce clinical symptoms which can be corrected by surgical operation.

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Figure 9
Patient standing in maximum lateral hip rotation

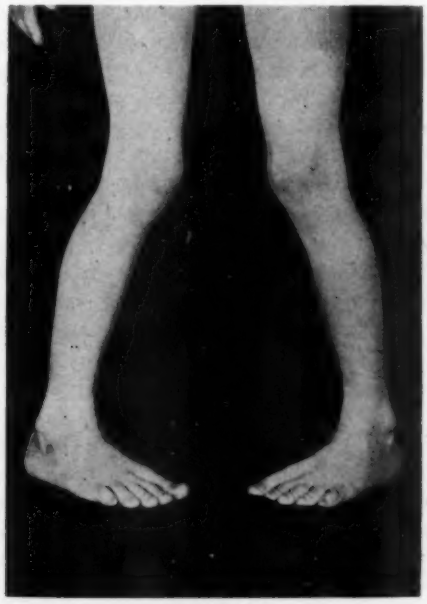

Figure 10
Patient standing in maximum medial hip rotation

DIABETIC COMA

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DIABETIC coma is the unique complication of Diabetes. It is the end result of untreated or completely uncontrolled Diabetes. In the strictest sense, it is a state of unconsciousness which accompanies the severe stages of diabetic acidosis. Today the term "diabetic coma" is more loosely applied, and it is necessary to set up certain arbitrary divisions. Any severe acidosis with a CO_2 combining power of 20 Vols. % or less is classified as coma. (At some institutions 15 Vols. % and at others 25 Vols. % is used as a dividing line.) The reasoning behind this is that before the discovery of insulin those patients who had a combining power above 20 Vols. % usually made a recovery, while those below did not.

Incidence of coma has decreased appreciably with the years since the use of insulin, but the death rate from it is still fairly high in many places. Three per cent is given by the Metropolitan Life Insurance Company as the over-all number of deaths due to coma.[1]

Pathological Physiology of Diabetic Coma

Diligent study of the chemical processes by which ketone bodies (acetone bodies) are formed from fatty acid has made it apparent that the former theories regarding their production must be modified. With newer, improved methods it is now possible to determine very small amounts of ketones, and minute amounts are found in the normal individual.

The old theory (Knoop Theory) was one of successive beta oxidation; that is, that the breakdown of fatty acids takes place in regular sequence by oxidation at the beta carbon atom with the production of a new fatty acid containing two less carbon atoms.

Now it is felt that there is multiple or alternate combustion and breakdown of the ketone bodies and that normally the body can utilize these incomplete breakdown products. But in the diabetic the breakdown is increased and the patient is unable to use the increased production. Therefore, a vicious cycle is set up with a depletion of CO_2, upset of acid base balance, dehydration, etc. Potassium and phosphate are also lost in great quantity with the ensuing diuresis.

A specific depletion of intra-cellular potassium which accompanies diabetic coma may be greatly increased by a diuresis artificially induced; that is, in treatment, when glucose is given, precipitating symptoms of respiratory failure.[2] Chlorides as well as fixed base are lost due to the vomiting and diuresis. The results listed below summarize the disturbed physiology occurring in diabetic coma:

1. Hemo-concentration and dehydration.
2. Depletion of both fixed base and the chlorides of body.
3. Lowering of the plasma CO_2 combining power.
4. Shift in the Ph to the acid side.
5. Depletion of glycogen stores in the liver and the muscles.

The dehydration and disturbances in *intracellular* electrolyte economy may be important in causing death, as they affect respiratory centers.

It has long been held that the utilization of carbohydrate exerts antiketogenic effect, and the primary aim in treatment is to improve the oxidation of glucose.

Etiology of Diabetic Coma

The factors may be tabulated as follows:

1. Too much food:
   a. Breaking of diet.
   b. Consumption of body tissues, as in undiagnosed Diabetes Mellitus, fever or Hyperthyroidism.
2. Too little insulin:
   a. None.
   b. Carelessness, ignorance.
   c. Discovery of Diabetes with coma.
3. Infection:
   a. Upper respiratory infection.
   b. Pneumonia.
   c. Carbuncle.
   d. Acute febrile disease.

The diabetic must be taught the dangers of overeating or of omitting insulin, especially in case of illness. So many times the patient will

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say, "I didn't eat, so I didn't take any insulin." Just recently I was attending a hospital patient with tuberculosis. She had a severe set back with hemorrhage from the lung, associated with loss of appetite. The hospital chart showed that the patient had not received insulin. I asked the nurse about it, and she looked unbelievingly at me and exclaimed, "Why *she* isnt' eating; we were afraid to give insulin!"

Many cases of severe Diabetes are not diagnosed until the patient is in coma. This leads to severe complications. This situation can be prevented by education and early recognition of the disease.

Symptoms and Signs

There are certain warning signs and some simple directions in which the patient and immediate family can be instructed in order to ward off coma. However, these can be vague and elusive to the doctor as well as the patient. Any of the following symptoms should be investigated: headache, anorexia, restlessness, weakness, listlessness, nausea, vomiting, drowsiness, painful and rapid breathing, or unexplained fever. A combination of the above should make one alert for coma, and patients should be taught the following rules:

1. Go to bed whenever indisposed.
2. Keep warm.
3. Call a nurse or some one to wait on you.
4. Call a doctor after testing the urine.
5. Drink a glass of liquid each hour—tea, coffee, broth, diluted orange juice, or oatmeal gruel.
6. Enema and fluids by rectum.
7. Appropriate amount of regular insulin on advice of your doctor.

Symptoms:

The onset of definite acidosis is attended by headache, malaise, nausea, vomiting, abdominal pain and generalized pain. It is also associated with thirst and polyuria.

Signs:

The patient lies in bed, is comatose, and may be moaning with pain. The skin is cold, dry and mottled; the face is flushed. The body temperature is subnormal; the pulse is rapid and weak; the blood pressure is low. The tongue is dry and coated and the eyeballs are soft.

Laboratory Findings

Blood sugar: Usually 440 to 550 mg. % or higher.

CO_2: 10 to 15 Vols. %.

Blood acetone: 100 to 190 mg. per 100 c.c.

Urine: ++++ sugar, acetone, diacetic acid (none in a renal block), showers of casts and albumin.

Hematologic change: high leucocytosis, (15,000 - 50,000).

Differential Diagnosis

There are a number of conditions to consider. The coma of a severe hypoglycemia can cause considerable confusion, although ordinarily the difference betweeen the two is quite great. If laboratory facilities are available, the differentiation is easy; if not, a concentrated glucose solution may be given as a trial. The history of onset is also very important.

Other causes of coma to consider are cerebral hemorrhage, uremia, meningitis, barbital poisoning, or toxemia of an overwhelming infection. Careful history, physical examination and laboratory work are essential in making a final diagnosis.

Prognosis

The prognosis depends upon the severity of the acidosis, the state and duration of unconsciousness, the age of the patient, the cardiovascular status and the complications that may arise—such as pneumonia.

An acidosis of short duration, twenty-four hours or less, will usually respond rapidly to treatment. An acidosis persisting for several days and culminating in unconsciousness will require more diligent treatment and may not respond satisfactorily for twenty-four to forty-eight hours after treatment has been instituted. A state of unconsciousness for twenty-four hours or more is very serious and it is usually this type of case which may succumb.

Children respond more readily to treatment and recover rapidly, principally because the duration of the disease is shorter and there are less apt to be complications. However, for the child who is poorly controlled, in mild acidosis frequently and in coma often, each succeeding coma may be a little more difficult to treat successfully.

Treatment

In order for the treatment to be successful, it must be instituted rapidly and energetically.

Hospitalization is to be desired, since adequate nursing care, laboratory procedures and special medications can be given without difficulty.

It is a good plan to have a routine program to follow for the treatment of coma; although, of necessity, the plan must be adjusted to the individual. The following suggestions are given as a program that has proved successful:

1. During the first hour after admission, the urine should be examined for sugar, diacetic acid, albumin, casts and pyuria. The patient should be catheterized if unable to void.

2. The blood should be tested for sugar and carbon dioxide content, obtaining a report within the hour. The white blood count and nonprotein nitrogen also are important.

3. A careful history should be obtained from the family to explain the cause of coma. A complete physical examination should be made to determine the state of unconsciousness, type of respiration, pulse rate, blood pressure and rectal temperature. One should also look for soft eyeballs, dry tongue, dilated stomach, cold and mottled skin, and impacted rectum.

4. One hundred (100) units of crystalline insulin should be given subcutaneously at once if the blood sugar exceeds 300 mg. % per 100 c.c., and if the blood carbon dioxide content is 20 Vols. % or less. The dose would be proportionately less in patients with recent onset of Diabetes or in young children. When the blood sugar is between 600 and 1,000, two hundred (200) extra units should be given; and where it is above 1,000, at least 300 units should be given in addition.

5. A gastric lavage with warm water, performed carefully, should be given to relieve the distention and remove undigested food particles.

6. Two thousand (2,000) c.c. of normal saline should be given intravenously; additional amounts can be given depending upon state of dehydration. Rapid administration must be avoided in older patients.

7. The patient should be kept warm, but burns to skin must be avoided.

During the second to the sixth hours, the administration of more insulin depends upon the severity of the coma, and occasionally it is necessary to repeat the first hour's dose in one hour. A repeat blood sugar should be taken at the end of three hours and 50 to 200 units of insulin given if the blood sugar is rising. As soon as the patient is able to tolerate fluids by mouth, 120 c.c. of broth, ginger ale, orange juice, tea or coffee should be given every hour. If the vomiting recurs, oral fluids should be stopped for twelve hours and the stomach lavaged again. During this period, an enema should be given to cleanse the lower bowel and relieve distention, and the urine should be checked for sugar and diacetic acid every hour. A record of the twenty-four-hour volume should be kept as well.

From the sixth to the twenty-fourth hour, definite signs of improvement should be noted. A repeat blood sugar should be taken and additional large doses of insulin should be given if the blood sugar continues to rise. If fall in blood sugar is satisfactory, insulin may now be given according to urine sugar tests every one to four hours as follows: (The urine test generally used is the qualitative Benedict method, although the Clinitest is equally satisfactory.)

Urinalysis Result	Red	Orange	Yellow	Green	Blue
Units of Reg. Insulin	20	16	12	0	0

On the second day, a diet of soft food may be given containing 100 to 150 Gms. carbohydrate, 50 Gms. protein, 50 Gms. fat. Administration of protamine zinc insulin should instituted, supplemented by crystalline insulin in small doses before meals as indicated by blood sugar and urine tests.

By the third day the patient should be returned to a standard diet for age and weight, with carbohydrate of 150 to 200 Gms., protein of 60 to 100 Gms. and fat of 60 to 120 Gms. daily.[3]

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THE USE OF CONTINUOUS CAUDAL OR SPINAL ANESTHESIA IN THE TREATMENT OF ECLAMPSIA

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A FACT basically recognized by all physicians is that the etiology of toxemia of pregnancy continues as one of the major unsolved problems in the fields of research and clinical medicine. The formidable significance attached to this subject is attested by the fact that even the best obstetrical centers throughout the country, such as The Margaret Hague Maternity Hospital, report their immediate maternal mortality for convulsive eclampsia as 10 per cent, and their fetal mortality as 33⅓ per cent.[1] Although these statistics are greatly reduced from statistics of two decades ago, a further reduction in mortality depends principally upon the detection of an unequivocally proved etiologic mechanism, from whence a specific treatment can be developed.

As has been so excellently summarized by Whitacre, Hingson, and Turner[2] in 1948, the presence of a generalized angiospasm in all toxemia patients, the etiology of which is undetermined, is in all probability one of the predominant factors causing the following changes in preeclampsia and eclampsia:

(1) Constriction of the arterioles of the ocular fundi,

(2) The fluctuations in blood pressure when the cold pressor test is applied,[3]

(3) The gradual hypertension of preeclampsia and the sudden rise in the blood pressure at the time of convulsions,

(4) Oliguria and anuria,[4]

(5) Intracranial hemorrhage and cardiac failure.

In concurrence with this angiospasm theory of mechanism it was decided in 1947 at this hospital to supplement the modified Stroganoff treatment of eclamptogenic toxemias with continuous caudal or continuous spinal anesthesia. The procedure was suggested through personal communications with Dr. Frank E. Whitacre who had had favorable though unpublished results with this method. The continuous caudal or spinal anesthesia produces a vaso-dilatation in the lower portion of the body tending to increase the renal output and decreasing the hypertension and resulting load on the heart which so often leads to intracranial hemorrhage, pulmonary edema, and cardiac failure.

Because we feel that our results with this procedure are indicative of its value in the treatment of eclampsia and preeclampsia, we wish to report in detail four of the cases which we have treated in this manner since 1947.

CASE REPORTS

CASE 1. A 25-year-old white primigravida whose past history was non-contributory, and whose expected date of confinement was November 18, 1947, had a relatively uneventful prenatal course with the exception of a slight ankle edema in the last trimester, and a slight elevation of blood pressure (140/90) one week before admission. At no time was albuminuria present, nor did the patient complain of headaches, photophobia, epigastric distress or other symptoms of impending toxemia.

Upon admission to the hospital at 8 a. m. November 14, 1947, the patient had been in active labor for six hours. Her membranes were intact, contractions were every three to four minutes, the cervix was dilated 6 cm., and the vertex was presenting at plus 1 station. Temperature was 98.0 F., pulse 74, respiration 18, blood pressure 120/80, and the general physical examination was satisfactory with the exception of a mild pedal and ankle edema. Labor proceeded in a normal manner, and at 3:46 p. m., under a terminal spinal anesthesia of 50 mgm. novacain the patient delivered a normal baby boy by episiotomy and low forceps. Shortly before delivery the blood pressure ascended to 150/90, but at the time of delivery it was 138/100. Obstetrical pitocin ½ c.c. was given intramuscularly after the baby, and ergotrate, gr. 1/320, was given intravenously after the placenta. At 4 p. m., during episiotomy repair, the blood pressure rose to 170/90, and the patient developed generalized convulsions and slight cyanosis, which lasted two and one-half minutes, and which presumably was relieved by 0.4 Gm. of sodium pentothal intravenously. The blood pressure immediately dropped, and at the time of leaving the delivery room at 4:25 p. m. was 138/78.

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One-half hour later the patient complained of a severe headache, was restless, her blood pressure was found to be 180/100, and ophthalmoscopic examination revealed flare hemorrhages. Since these symptoms precede further postpartum convulsions, continuous caudal anesthesia supplemented by sedation and hydration was decided upon as the therapeutic procedure of choice. Before the caudal equipment could be set up the patient had another convulsion, which was aborted by intravenous sodium anytal, and at 6:05 p. m. a continuous caudal was instituted using 1.5 per cent solution of metycaine. One hour after the initial injection the blood pressure had dropped to 100/70. By 8:40 p. m. the total urinary output since delivery had been 165 c.c., analysis of which showed 3 plus albuminuria, 1 plus acetonuria. At this time the blood pressure was 130/90, and the patient experienced a third convulsive seisure, which was controlled by 25 c.c. of metycaine through the caudal needle.

During the next five hours the patient voided an additional 960 c.c. of urine, rested quietly, and the albuminuria had progressively decreased to a trace on hourly specimens while the acetonuria had disappeared. During this time the blood pressure on several occasions became elevated as the metycaine effect waned, but repeated injections promptly caused it to drop, and the level of blood pressure could easily be regulated by varying the dosage of metycaine. In an attempt to keep the blood pressure fluctuations at a minimum, pontocaine was now substituted for metycaine as the anesthetic agent. Approximatetly thirteen hours after delivery, since the blood pressure was well stabilized at 120/8 0to 130/90, and since the total urinary output since delivery was 1800 c.c., analysis of which showed no albumin or acetone, the caudal needle was removed (a total of 1125 mg. of metycaine having been given) and therapy continued on sedation and hydration. During the next forty-eight hours the blood pressure ranged between 98/60 and 144/90, and during the first twenty-four hours of this forty-eight-hour period, the urinary output was 2540 c.c., while during the second twenty-four hours the output was 5600 c.c.

Laboratory studies on this patient, including blood urea nitrogen and nonprotein nitrogen determination, were withihn normal limits with the exception of a mild secondary anemia. Her hospital course was otherwise uneventful, and she was discharged with the baby on her ninth postpartum day in a greatly improved condition, and with a blood pressure which had remained fairly well stabilized around 112/80. When admitted to this hospital one year later for minor surgery, her blood pressure and urinalysis were normal.

CASE 2. A 30-year-old white gravida II expected the date of her confinement on May 29, 1948. The past history was negative and she had had a normal prenatal course until the morning of admission when she noticed increasingly severe headaches, vertigo, and photophobia. Examination at her physician's office revealed a blood pressure of 210/150 and 4 plus albuminuria. Although she was instructed to proceed immediately to the hospital, she preferred to return home where she experienced three generalized convulsions before she entered the hospital at 10 p. m. February 18, 1948.

At this time her temperature was 99.4 F., pulse 90, respirations 18, and blood pressure 170/110. She appeared acutely ill, was semicomatose and irrational; her skin was pale and moist; severe dependent, sacral, and flank edema were present. Ophthalmoscopic examination revealed definite arteriolar spasm. The fundus was at the umbilicus, vertex presenting but not engaged; the cervix was long and firm with no dilatation; fetal heart tones were 150 per minute.

Shortly after admission the patient had two more convulsive seizures, each of several minutes duration. For the first twelve hours after admission she was placed on conservative treatment of rest, fluids, sedation, and analgesia, which regimen produced no clinical improvement, and the urinary output was only 42 5c.c. Therefore, twelve hours after admission, a continuous caudal anesthesia was begun, but despite adequate anesthesia to any desired level by varying dosages of metycaine, the blood pressure remained constantly around 170/110. After nine hours of caudal anesthesia (325 mg. of metycaine having been given), the patient appeared less restless and her sensorium had cleared. However, because of the persistent hypertension and because no effective diuresis could be established and albuminuria continued at 4 plus, the caudal needle was removed and the patient was given a continuous spinal anesthetic by the Touhy catheter technic.[5] Twelve hours after the spinal was instituted the albuminuria decreased to 2 plus and remained that or below until discharge, and, although the total urinary output the first twenty-four hours was only 460 c.c., the output the second twenty-four hours was 2700 c.c., and for the twenty-four-hour periods thereafter varied between 1025 and 1900 c.c. The hypertension, however, failed to respond significantly at any time while under either continuous caudal or spinal anesthesia, indicating a pre-existing essential hypertension.

Fifty-seven hours after institution of the continuous spinal the patient was improved—effective diuresis had been established, albuminuria had disappeared, edema was definitely lessened—the spinal catheter was removed (a total of 259 mg. of metycaine had been given), and she was maintained on medical management.

During this time the patient had had irregular uterine contractions which had dilated the cervix to 2 cm. Inasmuch as she had two and one-half months gestation remaining, and because eclamptic attacks frequently recur, it was decided to induce labor in an attempt to prevent a future maternal mortality. Therefore, after routine medical induction, and after rupturing and stripping the membranes, she went into active labor, delivering six hours later a premature infant which expired shortly after birth.

Routine laboratory tests on this patient were negative with the exception of a mild secondary anemia. Consultation by an ophthalmologist on her second postpartum day revealed slight retinal edema of the right eye with old petechial hemorrhages. Her immediate postpartum course was uneventful and she was discharged on her eighth postpartum day with a negative urinalysis, blood pressure of 140/100, slight visual disturbances in the right eye, but otherwise in a normal state of health.

CASE 3. A 27-year-old primigravida, expected the date of her confinement to be March 22, 1949. She had had a negative past history and a non-contributory prenatal course with the exception of mild dependent edema for several weeks prior to delivery. At the time of admission to the hospital on March 17, 1949, she had been in active labor for eleven hours, and had noted no unusual symptoms. Her temperature was 98.8 F., pulse 80, respirations 20, blood pressure 158/100. She was experiencing contractions every eight minutes, appeared restless, pale, and acutely ill, and there was moderate pitting ankle and pedal edema. Examination of the abdomen revealed a term pregnancy, vertex presentation with good fetal heart tones; a rectal examination showed the cervix to be 1 to 2 cm. dilated with the presenting part at a minus 2 station. Immediate urinalysis revealed 4 plus albuminuria with numerous red and white blood cells.

It was agreed that the patient might be successfully managed on sedative and hydration therapy, which was instituted. However, seven hours after admission the blood pressure had risen to 210/114, and the patient had a moderately severe, generalized convulsive seizure lasting four minutes. A continuous spinal anesthesia was started immediately, using 1.5 per cent metycaine solution (at this time the patient was having regular contractions every two minutes and was 6 to 7 cm. dilatetd). Shortly after onset of anesthesia the blood pressure dropped to 190/100, and the patient went rapidly and uneventfully on to complete dilatation. About two hours after administering the anesthesia she delivered a normal living infant by means of low forceps and episiotomy. Three minutes after delivery, although the blood pressure had dropped to 130/85, she experienced another generalized convulsion of two minutes duration, which was easily controlled by additional metycaine, the blood pressure falling to 90/60.

The possibility of postpartum convulsions seemed imminent because of the occasional failure of the blood pressure to respond to repeated injections of metycaine and because of the persistence of the patient's severe headaches; consequently, the spinal catheter was left in place until the fourth postpartum day and the patient received repeated injections of metycaine. The first twelve hours after delivery the urinary outputp was 137 5c.c., albuminuria had decreased to 1 plus, and the blood pressure averaged 130/90. On the first postpartum day there was a marked diuresis, the total urinary output exceeding 5000 c.c., and the output the next five postpartum days averaged 2400 c.c. per day.

Laboratory examination, with the exception of a slightly elevated blood urea nitrogen, which three days later returned to normal, was essentially normal. In addition to intravenous fluids, sedation, parenteral vitamins, magnesium sulfate and narcotics for headaches, she was also given prophylactic penicillin, methionine, 500 c.c. of whole blood, intravenous amino acids, and a high-protein, salt-free diet. Her clinical condition improved steadily, and at the time of discharge with the baby on her ninth postpartum day, there was no edema, urirne was negative except for 1 plus albuminuria, and the blood pressure became fairly well stabilized at 130/98.

CASE 4. A 23-year-old white primigravida whose expected date cf oonfinement was April 22, 1949, had a normal prenatal course with the exception of a rising blood pressure and moderate transient dependent edema which had been present during the last month of pregnancy.

At the time of admission to the hospital in active labor at 8 a. m. April 16, 1949 she stated that she had had frequent headaches for the past few days, associated with some photophobia and epigastric distress. Her temperature on admission was 98.6 F., plus 80, blood pressure 168/108. Strong uterine contractions occurred every five minutes, and there was mild generalized and dependent edema in addition to a 1 plus proteinuria. Examination of the abdomen revealed a term pregnancy, vertex presentation, normal fetal heart tones, and upon rectal examination the presenting part was at a minus 1 station with the cervix completely effaced but not dilated. The patient was to be treated conservatively until the cervix began to dilate and the head to descend, whereupon a continuous spinal was to be instituted if the status of the

preeclampsia remained the same or became intensified.

Eight hours later, when the cervix was 5 to 6 cm. dilated, and the blood pressure was 156/122, it was considered advisable to begin the regional block, but inasmuch as the only trained resident-anesthetist was occupied elsewhere, it was necessary to continue the patient on the conservative regimen already in progress. Two hours later, when the blood pressure was 130/100, a terminal spinal anesthetic was given and the patient delivered a normal baby girl by means of episiotomy and low forceps.

Four hours after delivery she became comatose, had severe generalized convulsions, and was severely cyanotic. After resuscitative measures, her blood pressure was found to be 170/110, whereupon a continuous spinal anesthesia was begun immediately, and after the initial injection of 2 c.c. of 1.5 per cent metycaine solution the blood pressure dropped to 110/60. Thereafter, frequently repeated injections of ½ to 2 c.c. of metycaine kept the segmental level of cutaneous analgesia at thoracic six to eight, and also maintained the blood pressure around 130/90, as well as relieving the persistent headaches. The urinary output in the first twelve hours after delivery was only 75 c.c., but for the twenty-four hours following increased to 2200 c.c., and for the succeeding twenty-four hour periods averaged 1500 to 2000 c.c.

Fifty hours after anesthesia was instituted the spinal catheter appeared to be plugged, but diuresis had been effectively established and maintained; moreover, when the metycaine dosage waned the blood pressure was down to 152/100. The catheter was therefore removed (total dosage having been 622.5 mg.). In the event that oliguria or increased hypertension developed the anesthesia was to be reinstituted. However, during the next few days diuresis contuined, the blood averaged lower, and proteinuria decreased from 4 plus on the first postpartum day to negative on the second day.

Because of financial considerations the patient's laboratory examination and hospital stay were minimized, and she was discharged with the baby on her fifth postpartum day. By this time the headaches and edema had disappeared, the blood pressure was 140/100, and urinalysis showed 1 plus albuminuria and hematuria.

DISCUSSION

We have noticed on many occasions that intravenous ergotrate administered to the patient under spinal anesthesia has produced a considerable elevation of blood pressure, and therefore it, as well as pituitary oxytocic preparations, might be omitted in these cases as a prophylactic measure. With cases under continuous spinal anesthesia for long periods of time Dr. Whitacre has suggested that the type of anesthetic agent be changed from time to time; i.e. from metycaine to pontocaine to procaine or vice verse. He also advocates that the anesthesia be discontinued intermittently in the event that the patient remains under control for some hours until the blood pressure begins to elevate again or other untoward symptoms appear. We believe that a catheter should always be used in the spinal canal in preference to a needle, and that its end should always be directed upward. If the catheter is inadvertently directed downward no anesthesia, or a transitory type of "saddle block" anesthesia will ensue. It has been observed that when the catheter is left in the spinal canal for long periods of time that occasionally the tip of the catheter will become blocked by impingement against the meninges. In such cases it becomes necessary to either withdraw the catheter several millimeters and/or inject the anesthetic material with increased pressure from the syringe. Also, we have found in several instances where the level of anesthesia was not high enough to produce anesthesia to above the renal area, that by placing the patient in a slight Trendelenberg position with the head acutely flexed during forceful injection of the solution, a satisfactory level could be attained. When the anesthetic level is to or above the eighth thoracic level the kidneys and adrenals are denervated, and a bloodless phlebotomy of the lower two-thirds of the body is produced by a vasomotor relaxation.

SUMMARY

We have presented the case histories of four patients with eclamptogenic toxemia of pregnancy who were treated with continuous caudal and/or continuous spinal anesthesia supplemented by routine conservative medical management. Of the four patients, all were under 30 years of age, three were primiparas and one a multipara. In two of the cases the convulsive seizures occurred before delivery while in the other two they occurred during the immediate postpartum period. One patient was treated by continuous caudal, one by continuous caudal later replaced by continuous spinal, and two by continuous spinal anesthesia. The minimal length of time a patient was kept under continuous anesthesia was thirteen hours, while the longest administration continued for ninety-six hours. Mety-

caine was the anesthetic agent used in all cases except one where pontocaine was employed for a short time. The minimal caudal dose was 1125 mg., the maximum 3525 mg.; the minimal dosage under continuous spinal was 622.5 mg., the maximum 3810 mg.

Two cases responded with pronounced diuresis and a definite lowering of proteinuria within twelve hours after institution of therapy, while in the other two cases improvement was not definitely assured until twenty-four to thirty-six hours later. Three cases showed a definite decrease in hypertension during therapy, while only one case failed to show the drop in blood pressure shown by the others. Two patients were discharged with blood pressures slightly exceeding the arbitrary standards of 140/90.

In addition we have treated a considerable number of preeclamptics, as well as many cases of hypertensive cardiovascular disease in pregnancy with equal success.

We have not presented this paper with the idea in mind of this treatment being of definite curative value in all cases of eclampsia, but at least in our limited experience we humbly believe that it is the best treatment available at the present time.

Furthermore, we sincerely hope that this work will stimulate additional research and clinical surveys so that its potential value may be justly evaluated on a large statistical basis. In conclusion, we wish to warn, however, that this type of anesthesia is a powerful but dangerous therapeutic measure and should be used only by physicians who are thoroughly trained in its administration.

We wish to express our indebtedness and appreciation to Drs. John P. Hubbard, C. H. Kulaski, J. B. Kass, and Andrew A. Fulgenzi for their cooperation in permitting us to use their patients in this study, and to Dr. John E. Burnett for his active role in the treatment of the first two patients.

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Editorials

1950 Annual Meeting of the American Medical Association

San Francisco, California

Your Executive Secretary was privileged to attend the 1950 annual meeting of the American Medical Association held in San Francisco, California, June 26 through 30, 1950, with Headquarters in the historic Palace Hotel.

On Sunday morning, June 25, 1950, in company with Dr. Frank J. Milloy, Secretary; Dr. Robert S. Flinn, Councilor-at-Large; and Dr. Jesse D. Hamer, Delegate to the A.M.A., I attended the Seventh National (Grass Roots) Conference of the County Medical Society officers in the Gold Room of the Palace Hotel.

The meeting was called to order by A. M. Mitchell, M. D., Chairman, Terre Haute, Indiana; Gunnar Gundersen, M. D., member of the Board of Trustees of A.M.A., LaCrosse, Wisconsin, delivering the address of welcome. Worthy of note was a "TRUE and FALSE Questionnaire" on socialized medicine entitled: "What Do You Know for Sure?" distributed to each one in attendance, with the first twenty minutes allowed for answering. This quiz was conducted by L. Fernald Foster, M. D., Secre-

tary of the Michigan State Medical Society, Bay City, Michigan. While we will not discuss the results, you may rest assured your representatives fared very well with others in attendance and needless to say, this period of the program was both informative and instructive.

Three panel discussions were presented: (1) How to Organize a Community Health Council, Fred A. Humphrey, M. D., Fort Collins, Colorado, and Blair Holcomb, M. D. of Portland, Oregon, participating, the former taking the Affirmative viewpoint and the latter the Negative; (2) Providing special benefits through County Medical Society membership, Mr. Joseph Donovan, Executive Secretary of the Santa Clara County Medical Society, San Jose, California; and (3) How to set up a County Medical Society record system, Mr. Frederick W. Fagler, Executive Secretary of the Alleghany County Medical Society, Pittsburgh, Pennsylvania.

In the afternoon at this same location was held the Sixth Annual Meeting of the Conference of Presidents and other Officers of State Medical Societies. The meeting was called to order at 1:30 P. M., Clarence E. Northcutt, M. D., of Ponca City, Oklahoma, President, presiding. The first order of business included the presentation of annual reports of Executive Committees and the Secretary-Treasurer, John Farrell, M. D.; presentation of resolutions; appointment of Committees; and announcements.

Julian P. Price, M. D. of Florence, South Carolina, President-Elect and editor of the Journal of the South Carolina Medical Association, addressed the gathering on the subject: "Medicine's Greatest Gift." This address was followed by one delivered by A. Grant Clarke of New York City, President of the Medical Exhibitors Association, Inc. on the subject: "The Exhibitor's View of the Medical Convention." Harry Becker of Detroit, Michigan, Director of the Social Security Department, United Automobile-Aircraft-Agricultural Implement Workers of America CIO, spoke on "Labor Looks at the Problem of Prepaid Medical Care." His remarks were most controversial and a lively discussion resulted during the Question and Answer period from the floor. The Profession admirably rose to the defense of "The American Way" and challenged many of the inaccuracies and misstatements made representing the labor viewpoint.

L. C. Selusher of Jefferson City, Missouri, President of the Missouri Farm Bureau Federation, member of the Board, and Chairman, Rural Health Committee, American Farm Bureau Federation, spoke on the topic: "Better Health for Rural People—How to Get It." Baird H. Markham of New York City, Director of the American Petroleum Industries Committee of the American Petroleum Institute gave an inspiring talk on "American Democracy on Trial" and sparked for the defense spirited debate from the floor. The meeting was concluded following receipt of reports from the Reference Committee, Election of Officers and Executive Committee members, and the Presentation of Awards.

HOUSE OF DELEGATES

On Monday morning, June 25, 1950, at 9 o'clock, I attended my first meeting of the House of Delegates of the A.M.A., whose membership comprises a total of 198, in the Palace Hotel, followed by an afternoon's session. These two meetings were devoted primarily to the receipt of resolutions and appointment of Reference Committees to consider proposals presented by constituent bodies throughout the Nation. I was particularly impressed with the seriousness of this body's deliberations and the orderly processing of its important and diversified business. In one day alone over 70 pieces of business were transacted. The proceedings were well disciplined and the Speaker of the House, Dr. F. F. Borzell of Philadelphia, exhibited in masterly fashion, time and again, his familiarity with good parliamentary procedure and ability to cope with all situations arising. These sessions continued on Tuesday morning followed by the historic inauguration of the President Tuesday evening, with another full session Wednesday morning and afternoon. On Thursday afternoon the House reconvened for the election of officers and concluded its business. While I will not attempt to present a digest of the proceedings, all of which will be contained in an official report of the A.M.A. to be issued at a later date, I believe it is worthy of mention that the most controversial issue presented in my opinion was the report of the Reference Committee and subsequent adoption of a modified report of the Committee on Hospitals and the Practice of Medicine which denounces systems whereby hospitals hire salaried physicians for medical care and bills the patients for this care.

In the capacity filled to overflowing Gold Ball Room of the Palace Hotel on the evening of

June 27, 1950, at 6 o'clock (Pacific Coast Daylight Savings Time), broadcast Coast-to-Coast over two radio networks (ABC and MUTUAL), Elmer L. Henderson, M. D., of Louisville, Kentucky, new President of the A.M.A., truly stated is his opening remarks when he said: "In the annals of American medicine, this is an historic occasion." I shall never forget this momentous occasion. Flanked by Officers of the A.M.A. at an open meeting before its House of Delegates, Dr. Henderson took his oath of office and delivered his stirring inaugural address heard by millions of the American people. His complete address or excerpts therefrom will be published elsewhere in this Journal or possibly at a later date.

The San Francisco meeting was considered one of the most successful in the history of the American Medical Association. In excess of 20,000 persons were registered, of which more than 10,000 were physicians.

Just a word about the technical exhibits in excess of 300, and scientific exhibits approximating 150. Filling three floors of the Municipal Auditorium covering more than 100,000 square feet, they depict the great achievements of medical progress. The Scientific Sessions were likewise well attended, papers of National and International significance in excess of 300 being presented. They contributed much toward the success of the Ninety-ninth Annual Session (the one hundred third year) of the American Medical Association.

The 1951 annual meeting will be held in Atlantic City, New Jersey; the 1952 meeting in Chicago, Illinois, and the House of Delegates chose New York City, New York, as the location for its 1953 annual meeting.

Respectfully submitted,

Robert Carpenter,
Executive Secretary.

PHOENIX CLINICAL CLUB

The Case History in this discussion is selected from the Case Records of the Massachusetts General Hospital, and reprinted from the New England Journal of Medicine. The discussant under Differential Diagnosis is a member of the staff of the Massachusetts General Hospital. The other discussants are members of the Phoenix Clinical Club.

Massachusetts General Hospital
Case Report No. 31342

A forty-three-year-old longshoreman was admitted to the hospital because of shortness of breath.

He was well until six weeks before entry, when he first began to note shortness of breath and weakness of the back and legs on climbing stairs and ladders. At about the same time he began to have a dry cough, especially after exertion. He was told that he had bronchitis and was treated with medicines, which afforded no relief. He had had slight hemoptysis on two occasions. Three weeks before entry he became sleepless and orthopneic, requiring three pillows. He had frequent night sweats. A week later he noted pain in his knees and thighs, but no swelling or redness. He also experienced a mild low substernal pain. The dyspnea and weakness had progressed to such a degree that, at the time of admission, he could not dress himself. During the six-week period of illness he had lost 15 pounds; he had had to quit his job two weeks after the onset of his illness.

At the age of four he had scarlet fever. He had not had rheumatic fever or any other severe illness. Four years before his present illness he had submitted to an Army physical examination and had been found to be in excellent health.

Physical examination revealed a slightly pale man with rapid, shallow labored breathing and limited chest expansion. The tongue was dry and red. There was no clubbing of fingers or toes. The neck veins were large but not pulsating. Fine moist rales were heard all over the chest, anteriorly and posteriorly. No murmurs were heard. The heart was not enlarged. The aortic second sound was equal to the pulmonic. The abdomen showed some voluntary spasm, and the abdominal wall was "sore" from much coughing. Rectal examination revealed a normal prostate. Neurologic examinations were negative.

The temperature was 99°F., the pulse 100 and regular, and the respirations 28. The blood pressure was 118 systolic, 75 diastolic.

Examination of the blood showed a red-cell count of 6,100,000 with 17.0 gm. of hemoglobin. The white-cell count was 9700, with 54 per cent neutrophils, 12 per cent lymphocytes, 27 per cent

eosinophils, 6 per cent monocytes and 1 per cent basophils. The red cells and platelets appeared normal. The urine specific gravity was 1.030, and the p^H 5.2. The sediment contained 3 red cells and 1 to 3 white cells per high-power field, with an occasional hyaline or granular case. A stool was formed, dark and guaiac negative. Repeated smears of the sputum showed no tubercle bacilli. The serum nonprotein nitrogen was 37 mg. per 100 c.c., the calcium 9.0 mg., the phosphorus 3.0 mg., and the cholesterol 205 mg. The protein was 4.98 gm. per 100 c.c. with an albumin-globulin ratio of 1.8. The alkaline phosphatase was 16.6 units per 100 c.c. and the acid phosphatase 3.3 units. A cephalin flocculation test was negative in forty-eight hours. The carbon dioxide and chloride levels were normal. The blood Hinton test was negative. An electrocardiogram on the first day showed a normal rhythm of 100, a PR interval of 0.17 second, and a flat and diphasic T_2, and inverted T_3, a deep S_2 and inverted T waves in CF_2, CF_4 and CF_5. At the same time an x-ray film of the chest showed extensive density of a rather unusual pattern in both upper lung fields and in the central portions of the left lower lung field. The heart did not appear enlarged. The left diaphragm was slightly elevated, and there was mild scoliosis of the thoracic spine.

On the third day marked variation of pulse and heart rate and sounds were noted on expiration. The pulse slowed and weakened during inspiration. A diastolic gallop at the apex and an inconstant diastolic scratchy murmur over the pulmonic area were heard. The respiratory rate rose to 40 for twenty-four hours on the third day, then returned to the former rate of approximately 30. Sputum cultures for streptococci, pneumococci and fungi were negative. The venous pressure and circulation time were normal. Repeated tuberculin and trichinella skin tests were negative. Re-examination of the chest fluroscopically and additional films on the fourth day showed no appreciable change in the extensive increased density that involved both lung fields. Further x-ray studies one week after admission showed a normal intravenous pyelogram. A film of the abdomen showed no soft-tissue masses and a normal-sized liver and spleen. Throughout the bones of the pelvis there were scattered nodules of increased density. A gastrointestinal series with the patient in the horizontal position showed the esophagus to be normal. The mucosal folds of the stomach were greatly swollen. An area of rigidity was seen just below the cardia on the lesser curvature for a length of 8 to 10 cm. On the spot films there seemed to be a shelf at the upper and lower edges of this lesion. A Graham test faintly outlined a normal-appearing gall bladder.

On the eighth day the patient spat up 10 c.c. of blood, which he stated came from his nose. A gastric analysis after histamine showed 9.9 units of free acid and 17.5 units of tatal acid. The sediment contained numerous polymorphonuclear leukocytes and was negative for tubercle bacilli. Repeated stool examinations showed three plus guaiac tests. The hemoglobin was unchanged. Successive differential counts showed the eosinophils to vary from 9 to 24 per cent.

The patient became progressively weaker and more dyspneic. During the third week the temperature rose slowly to 101°F., the pulse to 120, and the respirations between 40 and 50. The urine and blood were unchanged. The peripheral lymph nodes were thought to be enlarged. Respiration became more labored, and without a terminal episode, the patient expired on the twenty-second hospital day.

DISCUSSION

Dr. Robert S. Flinn:

This is the story of a 43-year-old man who was ill for only six weeks prior to entry because of shortness of breath; weakness and loss of weight. On admission he had rapid shallow respirations and most of his symptoms seemed to be related to his respiratory system. He had a relative polycythemia of 6,100,000 with relatively normal white count except for the presence of 27 per cent eosinophils which varied in successive count from 9 to 24 per cent. His alkaline phosphatase was 16.6 units but otherwise his blood chemical examinations were not remarkable. Electrocardiographic tracing showed T Wave changes in Leads II, III and in the chest Leads, consistent with coronary artery disease or pericarditis. X-rays of his chest showed extensive density of a rather unusual pattern in both upper lung fields and in the central portions of the left lower lung field. The heart did not appear to be enlarged. The examination of the chest showed no appreciable change in the extensive increased densities that involved both lung fields. X-ray of the pelvis showed scattered nodules and increased densities. X-ray examination of the stomach showed a rigidity below the cardia on the lesser curvature for a length of 8 to 10 cm.

During the 21 days the patient remained in the hospital, he became weaker and more short of breath and one assumes that most of the symptoms were referable to his respiratory symptoms.

In considering a differential diagnosis, one must mention the various infections of the lung and here we must assume that the patient did not have pulmonary tuberculosis; lung abscess

or fungus disease. If you or I could have seen the x-ray plates, we might not have been so vague in our description. At any rate one must assume that this was not the typical picture of a metatastic carcinoma.

The presence of the eosinophilia is rather interesting and raises the question as to the possibility of an aleukemic leukemia; a lupus erythematosus; or a periarteritis. I see no evidence on which to make such a diagnosis, although periarteritis nodosus with involvement of stomach, heart, lungs is an attractive possibility. One also, I suppose, must consider the possibility of Hodgkin's disease and even Paget's disease.

If one attempts to make one diagnosis fit the entire picture, one must explain the changes in the stomach, in the bone, in the chest on the same basis. I can think of only one condition which might account for all these various manifestations and that, of course, is carcinoma of the stomach. This diagnosis is perfectly tenable in spite of the fact that the gastric analysis showed 9.9 units of free hydrochloric and 17.5 units of total assets, since, according to prevailing textbooks, free acid is present in the gastric content in about 40 per cent of cases, and after the injection of histamine, subcutaneously, may be found in rather high concentration—75 to 100 clinical units. Although bone metastases from carcinoma of the gastrointestinal tract are relatively uncommon, I am forced to explain the whole picture of the basis of carcinoma of the stomach.

Dr. Clarence B. Warrenburg:

The 43-year-old longshoreman who is our patient for today, was well until six weeks before admittance to the hospital. He was admitted because of shortness of breath. The rather complete clinical picture, along with the x-ray and laboratory findings that are reported on the first page of the protocol, makes one think of a number of diseases that this patient might have had. But on the second page there is recorded a finding that gives us a lead as to the diagnosis in this case. I am referring to the G. I. series report which states that an area of rigidity is seen in the stomach just below the cardia on the lesser curvature. This lesion is described as 8 to 10 cm. in length and there seemed to be a shelf at its upper and lower edges.

This lesion is undoubtedly a carcinoma of the stomach, until proven otherwise. The protocol does not give us the advantage of a gastroscopic examination. A flat plate of the abdomen shows no abnormalities except in this x-ray film there are revealed scattered nodules of increased density throughout the bones of the pelvis. An x-ray film of the chest showed extensive density of a rather unusual pattern in both upper lung fields and in the central portions of the left lower lung field.

No tubercle bacilli could be found on repeated tests of the sputum. The heart seemed normal except for a rapid rate. The liver, gall bladder and spleen were apparently normal. The blood Hinton test was negative and the neurological examination was negative.

The whole picture, with a few disconcerting exceptions, is compatible with the diagnosis of cancer of the stomach with extensive lymphatic and hematogenous metastasis to the lungs.

Those exceptions are:

1. There is no mention in this history of even a mild gastric distress.
2. The gastric analysis reveals free and total acid.
3. No anemia is present.
4. An eosinophilia is present but this is sometimes present in the advanced states of CA.

The findings that favor the diagnosis of CA of the stomach with metastasis to the lungs are:

1. The age and sex of the patient. Most CA of the stomach occur after age of 40. The condition predominates in the male sex two to one.
2. The x-ray finding of a lesion in the stomach.
3. The extensive density of a rather unusual pattern in both upper lung fields.
4. X-ray evidence of metastatic lesions in the bones of the pelvis.
5. The cachexia, pallor and fever.
6. The 15 pound loss of weight.
7. Occult blood in the stool; and the patient spat up blood on the 8th hospital day.
8. Enlarged peripheral lymph nodes.

MY DIAGNOSIS IS:

1. Inoperable carcinoma of the stomach with extensive metastasis to the lungs and to the bones of the pelvis.

Livingston and Pack in 1939 estimated that there were more deaths from gastric CA alone than from cancers of the lip, tongue, tonsils, cheek, pharynx, larynx, salivary glands, thyroid, breast (male and female), ovary, uterine cervix and croups combined. Moreover, they state that there has been no improvement in the salvage

from gastric cancer in the past 20 years. They estimate the definitive cures of unselected patients with gastric cancer at 2 per cent.

Yet gastric cancer is curable provided only that the patient reaches the surgeon with a respectable lesion. What is needed is a high index of suspicion of cancer in the patient who complains of a gastric disturbance which does not yield to conservative treatment within a period of a few weeks.

DIFFERENTIAL DIAGNOSIS

Dr. Wyman Richardson: The story is that of a relatively young man with an acute illness lasting four weeks.

Is it fair to look at the temperature chart? I was quite interested to know whether he had a high plateau type of fever or one that went up and down.

Dr. Benjamin Castleman: You may see it. It is not striking.

Dr. Richardson: The fever apparently started off gently and really never did get particularly high. It was not a septic temperature.

First of all I want to run over some of the findings. The primary symptoms were pulmonary, and there was slight hemoptysis early in the disease. I think that this is worth emphasizing. He had night sweats. This is also worth emphasizing. The urine was essentially negative. It contained a few blood cells but no albumin. Several sputum examinations were done; but the story was that of an unproductive cough and there is nothing to suggest that what he was bringing up was really sputum. The alkaline phosphatase was elevated, and this suggests bone disease with osteoblastic activity. The murmur in the pulmonary area could possibly have been due to pericarditis, or to pleuritis from the overlying lung, or it may not have been significant at all. Tuberculin tests were negative. Repeated tests with trichinella antigen were also negative. There was pus in the gastric contents, an observation not frequently made, perhaps because it is not looked for. Perhaps we should see the x-ray films at this time. I am interested in the lesion in the stomach and the appearance of the chest, as well as some of the bone lesions.

Dr. Milford D. Schulz: The films of the chest show extensive, symmetrically distributed mottled and stringy areas of increased density spreading into both lung fields. These go all the way out to the periphery of the lung fields and seem to be most prominent near the lung roots. It ushers in as possibilities a whole group of lesions that includes lymphatic spread of tumor, fibrosis, miliary tuberculosis, sarcoid and others.

Dr. Richardson: You never see an x-ray of the chest like that with periarteritis nodosa, do you?

Dr. Schulz: Not quite like it, although somewhat approaching it. The findings in this film in association with the increasing dyspnea make one think about lymphatic spread of tumor, especially in view of the areas of increased density evident in the bones.

Dr. Richardson: Let us look at the bones.

Dr. Schulz: There are many round areas of increased density in the pelvis that look like osteoblastic reaction to metastases. Similar bone lesions can be seen in lymphoma.

Dr. Richardson: Do you see areas of osteolytic activity?

Dr. Schulz: I do not believe so. I think that they are osteoblastic.

Dr. Richardson: With widespread tuberculosis in the bone, is there ostoblastic activity or is it mostly a destructive process?

Dr. Schulz: I have never seen it look like this.

Dr. Richardson: This is, of course, a miliary process.

Dr. Schulz: The fluoroscopist observed greatly swollen rugal folds. The films of the stomach show an area of rigidity along the lesser curvature, and this is quite constant on the spot films. No actual tumor mass is seen, but there is ulceration of the mucosa along the rigid part of the stomach.

Dr. Richardson: I want to play down that lesion.

Dr. Schulz: These swollen rugae are reminiscent of a case presented at this conference several years ago that proved to be merely gastritis with infiltration of the stomach wall.[1] Lymphoma of the stomach can also have this appearance.

Dr. Chester M. Jones: Would you be willing to say that this was not an extraluminal gastric lesion, but that it was in the mucosa?

Dr. Schulz: On the basis of these films I do not believe that one can tell.

Dr. Richardson: Now for the diagnosis. If one takes the eosinophilia seriously, what does one have to consider? I am not going through

all the possibilities, but some of them have to be considered seriously.

It do not know much about Loeffler's syndrome, but it has to do with a pneumonitis and a high eosinophilia. It is relatively acute and benign. I am not up on the literature, but in cases coming from the Mediterranean area I have seen mention of eosinophilia and chronic pneumonitis that lasted for a year or more. I do not believe that any of these cases had an acute fulminating process of this sort. I have seen one patient in this hospital who had an eosinophilia with lesions in the lung, a picture that suggested to me Hodgkin's disease. In the course of a year, however, all the lesions disappeared, as did the eosinophilia, and the patient made a spontaneous recovery. In the present case the disease seems to me to be too widespread and too quickly fatal to consider this group, and therefore I cross them out.

I think that one can make out a good case for periarteritis nodosa, whatever that may be. It is a clinical hodge-podge when one tries to make of it a clear-cut clinical picture. This case does not follow the pattern that one encounters in the asthmatic type. There is no peripheral neuritis, no mesenteric thrombosis, no renal symptoms, and only slight evidence of any actual coronary involvement. Furthermore, I do not believe that we see such a chest picture in the usual type of periarteritis nodosa. It seems to me that the course is too acute for a case of periarteritis nodosa, and in spite of the eosinophilia I am going to throw that diagnosis out. Also bone lesions would be extremely rare with periarteritis nodosa.

Trichinosis can be fatal. When it is, I believe that it would cause a typhoid-like picture, which this patient did not show. There may be central-nervous-system involvement in fatal cases. Two skin tests were negative. If one is going to pay any attention to such tests it seems to me that they should have been positive in this case. I am going to rule it out.

Tumor seems to me the logical diagnosis in this case. There is widespread disease involving bone with lesions that Dr. Schulz says are consistent with metastatic tumor of the bone. There is a lesion in the stomach, and one can logically say that this patient had a tumor of the stomach with widespread metastases, or possibly primary tumor in the lung, with metastases elsewhere. Eosinophilia with tumor involving the lung is sometimes observed. I saw a case not so long ago with eosinophilia due to bone metastases from a pancreatic tumor, but if one is going to suggest tumor as a cause for eosinophilia there should be an accompanying anemia and changes in the blood characteristic of widespread bone-marrow involvement. This man had no anemia; in fact he had a polycythemia, probably due to a certain amount of dehydration and possibly accentuated by some anoxemia, although he is not described as being cyanotic. I do not get the feel from this case that it was malignant tumor. It was rapidly fatal without any of the really good localizing signs or symptoms that one would expect with a rapidly fatal tumor. It is the kind of death that one might expect from acute leukemia. Nothing suggesting this was observed in the blood smear. Then we have the lymphoma group. It is rare to see a case as quickly fatal as this in any of the lymphomas, unless one includes acute leukemias, which I have already mentioned.

The whole picture to me is that of infection, and whereas I say that tumor is the logical diagnosis, I am going to throw that out also. It may be an infection that I know nothing about or that I have not thought of, in which case there is no use in talking any more. The only infection that I can think of that might produce such an acutely fatal illness is an acute miliary tuberculosis. Does miliary tuberculosis cause eosinophilia? I do not remember having observed it, although I have not seen many cases. That is one point against it. The tuberculin test was negative, but the tuberculin test is frequently negative in overwhelming infection by the tubercle bacillus. So we can allow that. The third point against it is this osteoblastic activity in the bone, which one would think ought to be osteolytic. I am not sure whether tubercles scattered throughout the bone marrow can "tickle up" the osteoblasts enough to lay down a little bone. I do not, however, feel satisfied with the diagnosis of tuberculosis.

The diagnosis of acute bacterial endocarditis ought to be entertained, but again there is no evidence of embolic phenomena, and the bone involvement does not seem to go with it. I am trying to make a soft bed for myself by saying that tumor is the logical diagnosis, but I am going to cross that out and say that this man

had a fulminating infection, probably miliary tuberculosis.

Dr. J. H. Means: Dr. Napier is here, and I have asked him to comment on the eosinophilia.

Dr. L. Everard Napier: My remarks will not bear any relation to this case, but I have been in the tropics, where we see a good deal of eosinophilia, much of which we cannot understand. We call it idiopathic eosinophilia. Frequently we find it running up to 60 to 70 per cent without any obvious reason. Another aspect of eosinophilia in the tropics is the high eosinophilic count in the normal population, as compared with the figures one sees in the textbooks that are based on experience in temperate climates. We have taken counts of so-called "healthy people" in India and found an average eosinophilic count of 14 per cent. A good deal of this is attributed to parastic infestation. That is not the whole story, however. When we exclude infestation we find an average eosinophilia of the lung or eosinophilic pneupresence of eosinophilia would not excite me the same way that it does you here. The one thing you think of is trichinosis or trichiniasis—it should really be called "trichineliasis" to be correct—but we do not think of that in India because we scarcely ever see it. We have seen eosinophilia of the lung or eosinophilic pneumonitis, but it is essentially a benign disease.

Dr. Jones: I saw this patient and thought that he probably had a lymphosarcoma originating in the stomach. The chest findings were perfectly consistent with such a diagnosis, and the x-ray film of the stomach showed a curious mucosal pattern which was like that of several cases of lymphosarcoma of the stomach that I have seen. On the basis of this trabeculation of the gastric mucosa and the rest of the story, that is, the chest findings and the bone lesions, I thought that lymphosarcoma secondary to a primary focus in the stomach was the logical diagnosis.

CLINICAL DIAGNOSIS

Lymphosarcomatosis.

DR. RICHARDSON'S DIAGNOSIS

Miliary tuberculosis.

ANATOMICAL DIAGNOSIS

Carcinoma of stomach with metastases to the regional lymph nodes, pleura, lung, pericardium, peritoneum and vertebras.

Lymphatic spread of pulmonary metastases.

PATHOLOGICAL DISCUSSION

Dr. Castleman: Both lungs were voluminous and filled the entire chest cavity. Scattered over both pleural surfaces was a yellow-white lacelike network, which reminded me a bit of the lacteals in the intestines when they are ballooned with fat. This network was bright yellow in places and formed a pattern of the entire lymphatic system of the pleura. It was very striking. This pleural process involved the entire pleura and extended on to the pericardium at the pleural reflection. This same lymphatic engorgement was seen throughout the parenchyma of both lungs. We, of course, thought of metastatic carcinoma and focused our attention on the stomach. Along the lesser curvature we found enlarged lymph nodes adherent to the pancreas, but the pancreas itself looked normal. Along this curvature we also found a slightly rigid area 2 to 3 cm. long that was only superficially ulcerated. The mucosal folds, however, were prominent, and this prominence is undoubtedly what was seen in the x-ray and led Dr. Jones to think of a lymphoma, because lymphoma does spread in submucosal tissues and elevates the mucosa. Of course, carcinoma can also spread in that way. These mucosal folds were high and could not be depressed so that we knew there was something, almost certainly a neoplasm, beneath the mucosa. Microscopically all the lymphatic vessels in the pleura and lungs were plugged with nests of neoplastic cells, which proved to be those of a differentiated carcinoma. The primary lesion was, of course, the rigid area in the stomach; it had extended throughout the submucosa to involve the serosal lymphatic vessels, the peripancreatic lymph nodes and the peritoneum. The vertebral bone marrow was grossly involved, and it is surprising that the red-cell count was high. Two or three other counts were taken, and all were in the neighborhood of 5,000,000.

Dr. Richardson: If one had looked at the smear I suppose one might have found some evidence of red-cell activity.

Dr. Castleman: This lung picture of lymphatic spread of metastases has been recently described by Dr. Mueller of the x-ray department and by Dr. Sniffen of this laboratory.[2] About 70 per cent of all cases of lymphatic spread of cancer of the lungs come from the

stomach; they usually occur in young persons, and the course is rapidly fatal.

Dr. Jones: I think that it cannot be stressed too much that miliary lesions in the lungs may be due to many causes other than tuberculosis. One of the most frequent is metastatic malignancy.

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TOPICS OF CURRENT MEDICAL INTEREST

RX, DX, AND DRS.

By Guillermo Osler, M. D.

This column recently resented the efforts of a well-known drug house to "help" the medical profession obtain a SYNTHETIC ADRENAL CORTICOID DRUG, for use with vitamin C in cases of rheumatoid arthritis. The reports were too controversial. . . In order to get an expert opinion, we have corresponded with a prominent arthrologist in Los Angeles. He has contributed to ARIZONA MEDICINE in the past, and has been of help in answering questions. . . . He says that a well-controlled study will shortly be presented at a medical meeting. The stuff is NOT valuable. (Actually the control cases, receiving distilled water, did better). . . . It is hard to see how the so-called successful series, even though small, could have been obtained.

Physicians are joining taxi-drivers in their rapport to a central office, VIA SHORT-WAVE RADIO. . . . Two hundred doctors in New York City will carry pocket-size receivers, with which they can receive calls from a telephone-answering service over a radius of 25 miles. . . . Each will have a number in code; the call goes through continuously; and the doctor answers by telephone—or keeps on playing golf.

The most logical and concise reason why socialized medicine is being pushed by the planners has been given by a Scot from London, Dr. Christopher Daniels. . . . "No nation in its right mind will accept Socialism or Communism. It must be introduced insidiously, through the emotions. BIRTH, DEATH, AND SICKNESS HAVE THE GREATEST EMOTIONAL APPEAL, and medical services are among the first to be attacked and taken over. A typical socialist trap is to bog the opposition down in arguments about the mechanism of the scheme, while the true meaning is being obscured. This situation is like polio—you don't know when you catch it, and by the time the paralysis is diagnosed it is too late."

The reports of GOOD EFFECTS from the use of curare ,procaine, cortisone, the antibiotics, etc., etc., are invariably followed by reports of TOXICITY. . . . No method seems to be as good as it looks at first, and only a few reach a favorable balance between success and hazards. . . . We should learn, and promise ourselves never to forget, that the trial of a substance almost always proceeds along certain regular paths, and that there are obstacles for all of them—even water.

A 1949 paragraph on WATER can now be modified . . . a method of distillation from sea-water by high speed centrifugal evaporation is being developed. Engineers in California say that the process will soon be practicable, and the water will cost about $20 per acre-foot. . . . This compares with $15 for the Colorado river water in that area—and who knows, the latter might become less plentiful?

Now and then the newest drug turns out NOT to be the best drug. . . . Eisele of Chicago reports in the March Wisconsin Medical Journal that aureomycin plus streptomycin is NOT as satisfactory or sulfadiazine plus streptomycin for BRUCELLOSIS. . . . The results are from a series of laboratory animals and cattle. Aureomycin is bacteriostatic rather than bacteriocidal, and the organisms promptly grow after use of the drug is stopped.

An oblique emphasis on progress in medicine is the publication of a SECOND edition of a ten dollar book on "BRUCELLOSIS" (H. J. Harris, for Paul B. Hoeber, Inc.). . . . Undulant fever has a brief history and a new therapy (as noted above) but there is a large backlog of human cases and a huge animal reservoir to be eliminated. . . The ARIZONA STATE VETERINARIAN is considering the use of more stringent controls over imported cattle. An occasional beast flares up with Bang's disease after being certified as negative when entering the state, and the loophole should be closed.

A recent note about CIVILIAN MEDICAL JOBS in the Canal Zone can now be revised to read—"and Japan, Guam and Okinawa." . . . The Department of the Army is seeking physicians in several specialties for one or two-year tours of duty as medical officers. Salaries vary from $6,400 to $9,500. The age limit is 57 years. Dependents are permitted, and living quarters are free or nearly so. . . . The applications go to Civilian Personnel Division, 1660 East Hyde Park Boulevard, Chicago 15.

One only has to wander a little bit among the sciences to get lost, or badly confused. . . . The spring issue of "Cathode Press," published by the Machlett Company, shows us a dim otherworld. . . . On page 2 we find a drawing labelled "schematic axial cross section of Super Dynamex Anode," and on page 6 an article entitled "A Ceramic Planar Power Tetrode.' . . . This inferiority feeling could be erased by inviting an engineer to a clinical-path. conference.

Dr. Ralph Waters, teacher of many current anaesthetists and inventor of many methods, must have a tolerant smile now and then as he tends his roses in Florida retirement. . . . Dr. Waters was the earliest professor of his subject, and headed the department at the Wisconsin General Hospital. . . . If he reads the hospital journals he will see pictures of the methods used to pipe OXYGEN to the bedside through three miles of copper tubing and 330 outlets in New York's Presbyterian Hospital. . . . The reason for his tolerance lies in the fact that he had oxygen piped into the W. G. H. in 1934, where it has been the accepted means of supply ever since.

Dr. John Lundy is chief of the Mayo Clinic section on anaesthesia, and also one of the great authorities in that field. Any pronouncement of his should carry special weight. . . . When he describes AN AUTOMATIC DEVICE TO REGULATE THE FLOW OF AN ANAESTHETIC as "fabulous," it should be just that. . . . The equipment contains an electroencephalograph, and the variation in "brain waves" determines the patients reaction. . . . The apparatus has actually been connected to the syringe which delivers an intravenous agent, and is said to control it well.

We have recently seen the report of a wonderful COMBINATION OF HEALING AGENCIES. . . . A Boy Scout was given a high award for his activities, which included hiking and other physical exercises. . . . He has been very active in civic affairs and in the Catholic Church. . . From birth until the age of ten he had a long series of operations to repair a clubbed and rotated foot. . . . The surgery was done through the aid of a Shrine Hospital, and he and his family are very grateful. . . . This sounds like Brotherhood—and it is also possible that some of the doctors didn't have ancestors on the Mayflower.

CARDIOVASCULAR CATHETERIZATION is a fashionable and valuable procedure right now. Quite a few articles are being published on its use in diagnosis, chiefly of congenital malformations. . . . It should be noted that the method is time-consuming, the pressures and blood analyses are difficult to obtain, and the data are hard to interpret. . . . Only a few clinics in the nation are capable of doing the job well—and that's the way it should remain for the time being.

We have blood banks, eye banks, bone banks, artery banks, etc., etc. . . . Now we have an "ICE BANK." . . . It looks suspiciously like a refrigerator, however, and is used in a hospital ward to produce ice in partially-filled pitchers for later use. . . . A 1950 ICE BOX.

Many unique answers to medical questions do not spring complete from the brows of professors. . . . A good example of this truism (if it is true) is THE HILLSDALE PLAN, started in 1948 by the Hillsdale County Medical Society of Michigan. . . . It was noted that 60% of the cancer cases treated in that area in 1946 involved the skin, breast, uterus, and rectum. Since all of these sites could be examined in the offices of physicians, without special equipment, it was arranged to make every office a cancer detection center. . . . Thirteen of the eighteen doctors in Hillsdale participate in the plan. A publicity campaign started the examinations at a high level, but the ball has continued to roll. . . . Several groups of patients are seen—women over 40 years who are seen by appointment; men or women of any age who request the routine examination; and patients who see their physicians recurrently for chronic diseases. . . . A specific history is taken, and the skin, mouth, throat, neck, breasts, heart, lungs, abdomen, genital organs and rectum are examined. The urine is tested, and a blood count is done if anemia seems evident. X-rays and biopsy are arranged if indicated. Re-examination is suggested at intervals of six months. . . . Many non-cancerous abnormalities have been found, and suggestions made for management. . . . The usual cost of the study has been $4 or $5. More than 1,500 original examinations were made in the first 18 months of the plan, and 54 cases (3.5%) of cancer were found, though only about 17% of the females over 40 in Hillsdale were examined.

A TESTING PROGRAM for substances which may be of value as replacements for natural hormones is gradually being formulated. The object is to find a compound which will replace a func-

tion of the hormone, and for which a test is available. . . . Examinations for the number of lymphocytes or eosinophiles, the sodium-potassium balance, etc., can be made on an animal. The adrenals (source of cortisone) are then removed, the substitute compound is then given, and the blood cell and chemical levels are noted to decide its efficiency.

The recent tendency has been to TREAT DIABETES with a "free" diet, combined with insulin, and little attention paid to the blood or urine sugar levels. . . . This is the path of least resistance for both the physician and the patient, and certainly the dream method for the patient. . . . The disadvantages are real, however. It is uneconomic, since more insulin is needed. It is hazardous to the pancreas, since hyperglycemia damages the islands of Langerbans. It may produce fatty degeneration of the liver. It also forms bad habits.

If a doctor applied for a job in the Arizona State Health Department, and then came to visit in Phoenix, the obvious conclusion would be that the two events were associated. . . . They could be, except for the notorious VERSATILITY OF PHYSICIANS. . . . A recent trip by Dr. R. Esmond Smith, member of the Los Angeles County Sanatorium Staff, and one-time medical missionary to Beirut in Syria, was made only in part to survey the Arizona job, and primarily to help found a parish of the strict and fundamentalist Scotch Reformed Presbyterian Church!

AN INTRODUCTION TO DIANETICS

LLOYD F. SMITH, M. D.,
Monrovia, California

You are walking down the street. A stranger stops you. "Pardon me," he says. "Are you Doctor White? Fine. I have discovered something so important that it ranks with the discovery of fire. With my discovery I can cure every psychosomatic ill, every neurosis, every psychosis. Are you interested?"

If that happens to you tomorrow, how do you expect to react? Wig-wag for a cop to grab the stranger until an ambulance can whisk him to a mental hospital? Laugh at this gigantic claim? Sit down with the stranger and listen to what he has to say?

That little story told above is no story out of a fairy book. It is something that has already happened. The stranger's name, L. Ron Hubbard. His address, Elizabeth, New Jersey. His claims stated in a book, Dianetics, published 1950 by Hermitage House, N. Y. The claims in the first paragraph above come almost verbatim from page ix, the very first page in his book.

Ordinarily we might like to dismiss such claims and go about our business. But the book Dianetics has hit best seller lists, and a foundation for Dianetics has been established in Elizabeth, N. J., or so I understand. Patients start to ask about it. Probably the best thing to do is to look into Hubbard's theories and see if we can understand part of them.

To explain his system of Dianetics, Hubbard has coined many new terms. His glossary defines thirty-five such new words, and throughout his book he adds possibly another fifteen or more. At least four words we might examine here—dianetics, analytic, reactive, engram.

Dianetics, says Hubbard, comes from a Greek word "dianoua," which means "thought." He uses the word to cover his new "science of mind" or Dianetics. At times Hubbard talks as if we have two minds, again as if we have one mind with two or more arbitrary parts. The terms analytic mind and reactive mind come in for constant mention.

Analytic mind is the mind or part of the mind we all admit and feel rather proud of. By it we are aware of "I," we remember things, we reason and compute. It bears marked similarity to what psychiatrists call the "conscious mind."

Reactive mind is the mind or part of the mind that was hidden, unknown until Hubbard discovered it. It has some resemblance to the "subconscious mind" of psychoanalysis. However, the reactive mind never sleeps. It is always there to plague us and to drive us into acts that we know, with our analytic mind, may be foolish, dangerous, criminal and so on.

Engram is the thing that makes our reactive mind such a nuisance. Hubbard defines the engram both as any period during which the analytic mind shuts down, and the total content of that period which the reactive mind records.

It works something like this. The analytic mind functions more or less all the time except under certain circumstances. Injury, illness and drugs seem to be three things that shut down the

analytic mind. With the analytic mind shut off, the reactive mind comes into its own and makes a record of everything that goes on. So, between the analytic and the reactive mind we have a continuous record stored within us, a record of our entire life. When Hubbard says entire, he means entire, from conception to death.

If only our analytic mind could work in peace, all would be well. But our analytic mind is not as rugged as our reactive mind. If we crash our auto and get stunned for a moment, out goes the analytic mind, in comes the reactive mind to record what goes on. Should we be very sick, say with a high fever and delirium of pneumonia, again the reactive mind takes over. Or drugs may do it, such as a big dose of barbiturate, or alcohol, or ether during an operation.

Hubbard does not locate this reactive mind, nor does he even identify it with the nervous system. He does not assign it our ordinary "sensation," but rather says it records on a "cellular basis." It acts like a perfect sponge to soak up everything going on. Everything the reactive mind records becomes the engram. The engram contains all the physical and emotional pain of that period of unconsciousness, everything, too, that bystanders say or do while the analytic mind shuts down.

Most dangerous of all seem to be any words spoken while we are in this state or stupor or apparent unconsciousness. The reactive mind has trouble with words. Having a submoron mentality, our reactive mind takes everything literally. It has no sense of humor, no ability to enjoy simile, metaphor or allegory.

Recall the famous story of a station agent who could not distinguish the kinds of pigs. To that Irishman for all time "Pigs is pigs." He could not distinguish between the barnyard hog and the laboratory Guinea pig. As a result he was swamped by Guinea Pigs, while he tried to figure out if there might be a time when "Pigs is pigs" does not apply. The reactive mind thinks in similar terms.

For instance, we may do harm whenever we talk in the presence of some one injured, very ill, or under the influence of drugs. If we visit a child delirious with meningitis, we should not say a word. If we say, "It's finished," meaning that you are ready to leave, the child's reactive mind may record that and use it to finish up the child's days on earth. Our reactive mind, says Hubbard, "thinks" only in terms of identity, our analytic mind in terms of difference and similarity.

Engrams, or what happens in moments of shut down by the analytic mind, lie at the root of all evil. Consider a farm boy riding a horse. He falls off. His head strikes the ground. For a time he is "unconscious," or at least his analytic mind shuts off.

If the boy falls off the horse out in the fields, he is lucky. With no human near, he has not too much to worry about. His engram during his time of "unconsciousness" will contain chiefly his bodily pain, plus such things as the hoofbeats of his horse.

On the other hand, suppose he falls off as he rounds the corner of the farm barn, just back of the house. Mother and father may hear him fall. They run out. Mother screams, "My poor baby. He's dead." Father looks disgusted, "Fool kid. Get up."

According to Hubbard the reactive mind takes all this in, records everything. Worse still, it takes it literally. Buried in that engram forever may be the conflict from orders to the boy to be "poor baby," "dead," "fool," "get up."

Not that this all follows at once. The engram has to be "keyed-in" at some later date. "Keying-in" an engram seems to be the restimulation of all or part of the content of the engram by a somewhat similar incident. Suppose in later days the farm boy moves to a city. He is playing tag in a vacant lot and hiding in the weeds. A cop has been called to drive the kids out of the lot, since a neighbor objects to the shouting incident to the game of tag.

So the cop comes upon the boy hiding in the weeds. The cop feels grouchy. "Fool kid. If you're not dead, get up." The words "fool," "dead," "get up" all "key in" the previous engram, and begin their deadly work. By engrams we become slaves to chance words. The "Tyranny of Words" so ably presented by Stuart Chase appears mild by comparison to the "Tyranny of Engrams."

All our lives we go on collecting these engrams, dozens, or hundreds of them. Engrams enslave us, make us neurotic, drive us insane. Behind every act we disapprove of in others, from murder to war, lies one or more engrams. Instead of blaming ourselves for what we dis-

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RESEARCH IN THE SERVICE OF MEDICINE **SEARLE**

like most about ourselves, we should blame our engrams. Hubbard insists strongly that he studies in engrams not what the individual does, but what has happened to or been done to the person in engrams.

If this be true, then our prime care should be to prevent engrams when possible. For this we preserve absolute silence around any one injured, ill, drugged. The engrams we already have we must get rid of. Should we once get rid of all our engrams, we become a "clear," an ideal person, better than any one ever seen on this earth up to now. Compared to any one today who thinks he is "normal," the "clear" would be a superman.

Through Dianetics, Hubbard says he can get rid of, or erase, or release the engrams. How? We must find a way to go back and re-experience the whole incident when the engram occurred, when we were "unconscious. Hubbard seems to insist that we do this by "returning," and he emphasizes that he does not mean mere recalling or remembering. Just what he means seems hard to grasp, and he admits that the only way to know is to follow his directions. For the moment his "returning" seems to be the attempt to vividly recall in minute detail all that happened during the moment of "unconsciousness," just as if it were happening again.

By his method we "return" to the engram, and keep going over and over it. Sooner or later the engram will erase or disappear, and be filed again under standard memory of the analytic mind. Gradually, as engram after engram lifts, we come into the fullness of our human potential. The drag of our reactive mind stops. Our analytic mind comes into full play. We are "clears" beyond the dreams of Zarathustra who spoke to Nietzsche.

What makes all this harder seems to be that engrams which matter most come before birth. More, engrams may begin at or shortly after conception itself. How then can we hope to touch those events and get rid of them? Easy, says Hubbard. To "return" to your prenatal life and re-experience what happened to you during prenatal life we find simple by his method of reverie.

When I first bumped into this claim that we could at will study our prenatal days, from conception onwards, I pulled up with a start. It made me feel the same way I did my first day as interne in a mental hospital. The first patient I met inside the locked door approached. I smiled and nodded to him and said, "Hello. And who are you?" That patient looked at me coldly, and spat at me one sharp word. "God," he said, and stalked on. Since then, what other people say does not startle me as much.

So Dianetics says that we must go to our prenatal days and re-experience over and over again the engrams that happened then. But you say that the fertilized egg will not get injured, very ill, or drugged. Not so, says Hubbard. The fertilized egg gets hurt during intercourse, bumped hither and yon. Mother pokes at it with whatever she has handy to try to abort it. When she gets constipated and bears down hard in her attempts at a bowel movement, baby again gets squeezed and hurt enough to produce an engram.

Douching bothers baby. Bumps of mama's belly on the kitchen sink do the same. Birth itself becomes a severe engram, as the uterus and abdominal muscles push and shove us towards the outer world. Baby changes from the picture of folk-lore as a "dear little thing, cuddled close beneath mother's heart" to a much abused child. During any or all of such types of "injury" to baby, every thing that goes on around it records on the engram bank.

If mother talks to herself, beware. As she strains at her stool, or fiddles with her slippery ellum to try aborting the baby, she may talk and the words register on the fetal engram bank. This is no attempt at sarcasm or humor on my part. I merely am trying to report what Hubbard says in his book on Dianetics.

How then shall we be cured of our engrams? We have an auditor who helps us to get well, who guides therapy. To show how an auditor works, I quote from pages 199 to 202 of Dianetics. In these quotes everything is taken literally from Hubbard except the few parts in parentheses which are marked with a (*).

"The patient sits in a comfortable chair or lies on a couch in a quiet room, where perceptic distractions (* i. e. sensations*) are minimal. The auditor tells him to look at the ceiling."

"Auditor: Look at the ceiling. When I count from one to seven your eyes will close. You will remain aware of everything which goes on. You will be able to remember everything that happens here. You can pull yourself out of anything which you get into if you don't like it. All

right (slowly, soothingly): One, two, three, four, five, six, seven. One, two, three, four, five, six, seven. One, two, three, (patient's eyes close and eyelids flicker) four, five, six, seven. (Auditor pauses.) (*At this point the auditor inserts the 'canceller'*.) (. . the canceller is vital . . . a contract with the patient that whatever the auditor says will not become literally interpreted by the patient or used by him in any way.)

"Canceller. . . . In the future when I utter the word Cancelled, everything which I have said to you while you are in a therapy session will be cancelled, and will have no force with you. Any suggestion I have made to you will be without force when I say the word Cancelled. Do you understand? All right, let us go back to your fifth birthday . . . (*or any other time, such as the earliest moment of pain in your prenatal life*)."

"(Work continues until the auditor has worked the patient enough for the period.)" (*Hubbard says the time spent in each therapy session averages two hours.*)

(*At the end of the time the auditor says*) "Come to present time. Are you in present time? (Yes.) Cancelled. When I count from five to one and snap my fingers you will feel alert. Five, four, three, two, one. (Snap.)"

What Hubbard calls "work" in the "dianetic reverie" consists of "returning" to the engrams and going over and over them until they erase, or disappear, to be refiled under standard memory in the analytic mind.

This reverie looks like a mild form of hypnotism. Not so, says Hubbard, since in hypnotism the patient goes to sleep. Also hypnotism suggests things to a patient while dianetics "returns" him to a point of time. How the auditor's words like "Return to . .", "Contact that more closely . . ", "Continue to repeat . . " and so on fail to equal suggestion is a fine point here. Somehow I fail to see the distinction.

As with any book that is meeting the ovation such as Dianetics gets in some quarters, we can not say the last word. Some people believe every word written in the book. The few persons whom I have personally known thus far, who have started Dianetic therapy, enthuse over it. Others laugh at the whole idea as too absurd to talk about.

Has Hubbard found something? Even if the patients do not "return" as literally as Dianetics seems to claim, what actually does happen in dianetic reverie? I do not know. My own attitude resembles that of Montaigne as regards miracles. Montaigne said that even if he saw a miracle he would not credit his senses. And dianetic looks to me like a miracle.

As in so many things, argument is futile. Time will sit in judgment here as always. At least Hubbard is no piker. He has stuck his neck out, way, way out. "With the techniques presented in this handbook . . . (an) intelligent *layman* can *successfully* and *invariably treat all* psycho-somatic ills and inorganic aberrations." By "inorganic aberrations" he seems to mean all neurosis and all psychosis.

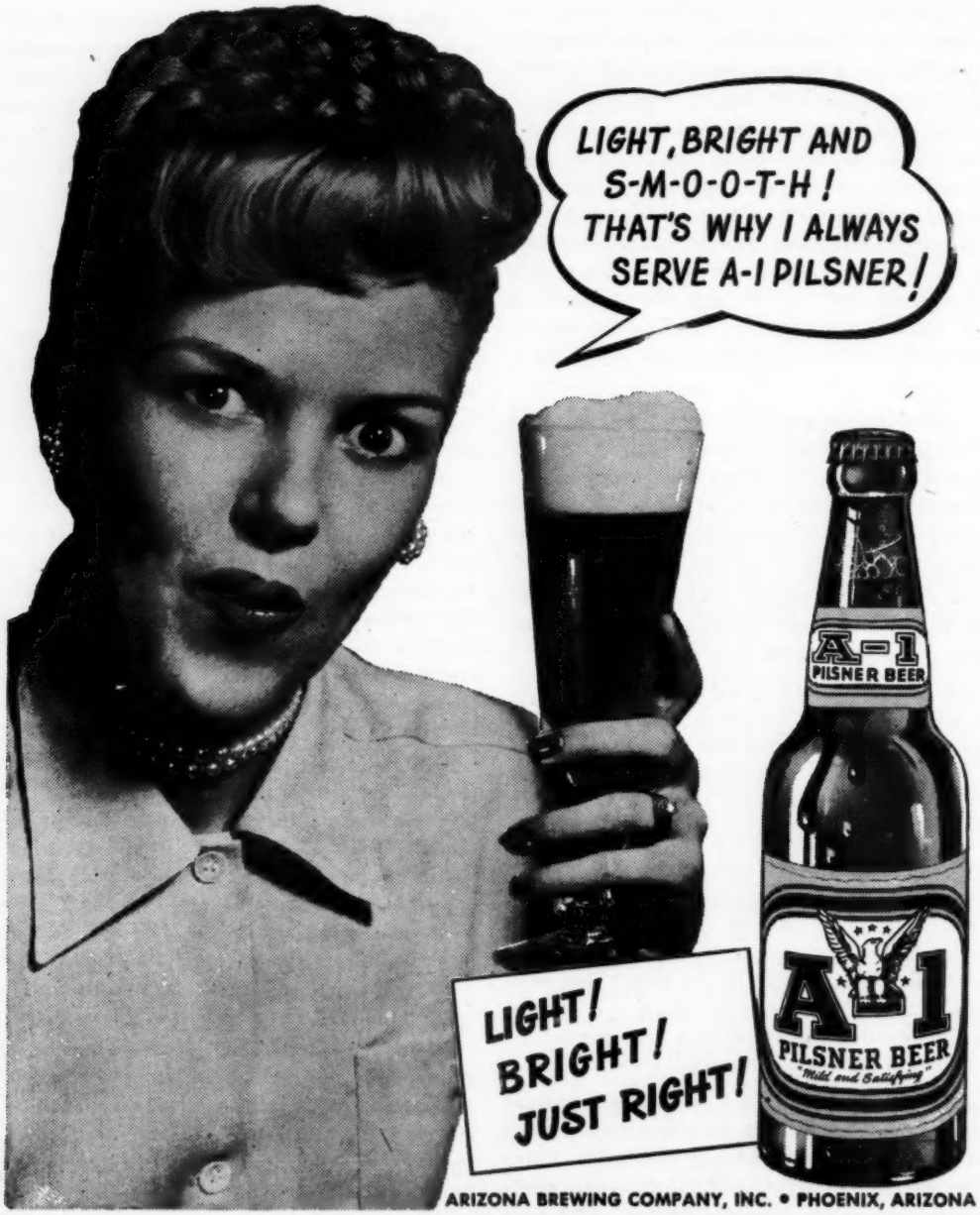
Again Hubbard does not try to conceal anything. He lays all his cards on the table and challenges us to experiment with it. He appeals to our fair play to judge not, but experiment. The future of dianetics may be worth watching. The book itself has hit best seller lists, and patients want to know about it. The Dianetic Foundation is said to be training professional auditors and branching out. In the end there may be something that Dianetics has to offer to modern psychotherapy. We can only wait and see.

You may like to ponder the two key sentences in the book. On page ix the book begins, "The creation of dianetics is a milestone for Man comparable to his discovery of fire and superior to his invention of the wheel and arch." I know of no boast in history to surpass that. The book ends on page 435 with a bit of wry humor and advice to those who try out its methods, "Good hunting." That is, I presume, "good hunting" for engrams.

A couple of decades ago I came on some doggerel somewhere, which in my vague recollection goes like this in part:

"It kin be jist so,
I dunno.
Jist so, coudda bin.
Then agin . . .?"

134 South Myrtle.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PERSONAL NOTES

Dr's. PAUL CASE and **ARCHIE CRUTHIRDS,** Phoenix, attended the Pacific Coast Oto-Ophthalmological Society in San Francisco, California, May 29th.

WALTER CRAIG, Phoenix attorney, concluded the series of talks on Medical Jurisprudence, before the Staff of Good Samaritan Hospital, Phoenix, May 22nd. Short papers were also presented by **DR. CHARLES VAN EPPS,** Phoenix, on "Achondroplasia of the Fetal Skull;" by **DR. ROBERT CUMMINGS,** Phoenix, on "Spontaneous Rupture of the Bladder;" by **DR. L. C. McVAY,** Phoenix, on "Spontaneous Rupture of the Uterus," and by **DR. HENRY G. WILLIAMS,** Phoenix, on "Rupture of the Gallbladder."

The June Staff meeting of St. Monica's Hospital, Phoenix, was held on June 13th, and was devoted to a symposium on "Prostatectomy." **DR. PAUL SINGER,** Phoenix, spoke on "Perineal;" **DR. R. H. CUMMINGS,** Phoenix, on "Transurethral;" **DR. L. C. WORMLY,** Phoenix, on "Suprapubic," and **DR. L. L. STOLFA,** Phoenix, on "Retropubic Prostatectomy."

DR's AUDREY G. URRY and **WALLACE A. REED,** Phoenix, and **DR. C. CLARK LEYDIC, Jr.,** Tucson, have been certified as specialists in anesthesia by the American Board of Anesthesiology.

DR. JESSE D. HAMER, Phoenix, presided at the Rocky Mountain Regional Conference on Voluntary Health Insurance in Salt Lake City, Utah, May 20th. He is a member of the American Medical Association Council on Medical Service. **DR. CARLOS CRAIG,** Phoenix, attended as President of the Arizona Blue Shield.

DR. J. P. WARD, Phoenix, State Director of Public Health, spoke before the New Mexico Public Health Association Conference in Raton, New Mexico, May 15-18, on "A Health Officer Looks at a Mental Hygiene Program."

DR. JOHN GREEN, Phoenix, took part in a symposium on the "Temporal Lobe" at Atlantic City, New Jersey, in early June. He spoke on effects of surgical removal of portions of the temporal lobe. This was a meeting of the American Electro-Encephalographic Society.

The June Staff meeting at St. Joseph's Hospital, Phoenix, was held on June 9th and was devoted to a symposium on "Glaucoma" by **DR's. PAUL CASE, O. W. THOENY, P. McFARLAND, H. FRENCH** and **P. LOVELESS,** all of Phoenix.

DR. M. L. SUSSMAN, Phoenix, spoke on "Congenital Heart Disease" to the Staff of St. Mary's Hospital, Tucson, and on "Non-Tuberculor Chest Lesions" to the San Diego County Medical Society, June 13th.

The June Staff meeting at Good Samaritan Hospital, Phoenix, was held on June 26th, and papers presented on "Skin Manifestations in Systemic Diseases" by **DR. HARRY CUMMING,** Phoenix. **WALTER, E. LOX, Ph. D.,** Phoenix, spoke on "Non-Pathogenic Protozea as Causes of Amebiasis," and **DR. GEORGE McKHANN,** Phoenix, spoke on "Treatment of Amoebic Infestation."

DR. WILLIAM H. OATWAY, Jr., Tucson, has been appointed Superintendent and Medical Director of the LaVina Sanatorium, LaVina, California.

DR. MARCY L. SUSSMAN has been appointed Clinical Professor of Radiology at the University of Southern California. He will act in a consulting capacity in the research program.

Arizona Teenagers Recognized

The Bureau of Health Education of the American Medical Association, under the direction of W. W. Bauer, M. D., has been developing throughout the Nation, a series of transcripts on Teenage Health Problems. They will be released to broadcasting stations through local and State Medical Societies late this year.

The program is arranged featuring groups of high school students in various cities of the country participating in informal discussions. The topics have been chosen from questions most often asked by teenagers themselves, according to field work and research done through A.M.A.'s School Health Division. These questions are related to social and emotional adjustments as well as purely physical problems.

Only thirteen states will be represented, providing thirteen recordings for a thirteen week—thirteen minutes each broadcast. Phoenix, Arizona is fortunate in being chosen to represent the Southwest area.

On Friday, July 7, 1950. the stage was set. At 10 A. M. in the broadcasting studios of radio station KOY—Phoenix, assembled the cast, including four representative teenagers previously selected from among the local high school

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student bodies, Dr. Bauer and the Producers, Marshall-Hester Productions of New York, represented by its principals; the subject: "Smoking and Drugs." Following the transcription, it was reported that the people of Arizona might well be proud of its "teenagers" judging from their informed and intelligent response throughout the discussion.

Following the transcription, Dr. Bauer and the Producers, Mr. Marshall and Miss Hester, were luncheon guests of the Arizona Medical Association, represented by Dr. Frank J. Milloy, Secretary; Dr. Robert S. Flinn, Councilor-at-Large, and the Executive Secretary, joined by members of its Auxiliary, Mrs. Benjamin Herzberg, President; Mrs. George S. Enfield, Vice-President and Program Chairman; and Mrs. Jesse D. Hamer, Parliamentarian of the State and Officer-Director of the National Organization.

Health education problems and proposed projects were considered.

Respectfully submitted,

Robert Carpenter,
Executive Secretary.

National Education Campaign American Medical Association

Chicago, June 12.—The American Medical Association, by unanimous action of its Board of Trustees and Campaign Coordinating Committee, has given the final "go ahead" signal for a Nationwide advertising program which will include three principal media — newspapers, magazines and radio — as a new phase of its National Education Campaign in behalf of Voluntary Health Insurance and against socialized medicine.

Clem Whitaker and Leone Baxter, directors of the A. M. A.'s National Education Campaign, announced authorization of the advertising program today and stated that the ad campaign will be launched in October.

A total advertising budget of $1,110,000 has been approved by the A. M. A. Board of Trustees, it was announced, with $560,000 allocated to newspapers, $300,000 to radio and $250,000 to National magazines.

The newspaper advertising schedule calls for blanket-coverage of every bona fide daily and weekly newspaper in the United States—approximately 11,000, in total—and the copy is scheduled to run during the week of October 8th. Newspapers in the territories of Hawaii and Alaska will be included in the schedule. The space reservation will approximate 70 inches (probably a 5 x 14) in all papers.

About 30 of the leading National magazines, and a score of advertising trade publications, will be included in the magazine ad program.

The radio advertising program calls for an intensive "spot announcement" campaign, utilizing time on some 300 radio stations, covering every State and Hawaii and Alaska.

The magazine and radio campaigns also are scheduled for October.

"This is not an institutional advertising program," said the Whitaker & Baxter announcement.

"The American Medical Association is embarking on a Nationwide advertising program for two reasons. First, it is determined to aid in every way possible in increasing the availability of good medical care to the American people through the medium of Voluntary Health Insurance. In that respect, the advertising copy will be designed to make the American people 'health insurance conscious' and to encourage the extension and development of prepaid medical and hospital care as a means of taking the economic shock out of illness. Second, American medicine is determined to alert the American people to the danger of socialized medicine and to the threatening trend toward State Socialism in this country.

"The ad copy, in part, will be designed to sell a commodity, Voluntary Health Insurance, but not any particular brand or plan. The individual will be encouraged to secure sound coverage in the plan which he feels best suits his individual needs. In its second aspect, the ad copy will be used to mobilize public opinion in support of a basic American idea—the principle of individual freedom, as opposed to the alien philosophy of a Government-regimented economy."

It was announced that the following advertising agencies have been selected to handle the buying of space and placement of copy:

Lockwood-Shackelford Company for newspaper and magazine media, with Homer J. Buckley & Associates, Inc., affiliated with them.

The Russell M. Seeds Company in the radio field, with the Harry M. Frost Company associated with them.

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Arizona Blue Shield Blue Cross

It cost short term hospitals an average of $14.33 to take care of a patient for one day during 1949, it is reported by the American Hospital Association, parent organization of the ninety Blue Cross Plans. The quoted figure, based on the Association's annual survey of more than 6,000 hospitals in the United States for the year ending last September 30, is up from the $13.09 it cost in 1948 and from the $11.09 figure for 1947.

The Association's report showed a $13.94 per patient day cost for Arizona short term hospitals in 1949, thirty-nine cents below the national average.

The income of the nation's general hospitals as a whole came nearer to meeting costs of care provided than it did in 1948, the survey revealed. However, the income per patient in all hospitals was $2.59 a day less than it cost the average hospital to care for him.

"Hospitals' healthier financial condition during 1949 was reached in a period when many more patients were cared for, and they more nearly paid the cost of that care," said George Bugbee, executive director of the American Hospital Association.

Patients in *short term non-profit hospitals* paid $138,129,000 less than the hospitals' expenses, the American Hospital Association reports. This deficit amounted to $1.56 for each patient daily. Money to pay for this came from sources other than patients. Some of these sources were endowments, contribuations, and payments by governments for care of people who couldn't pay.

Hospitals' expenditures were up half a billion dollars in 1949 over 1948, to a new high of $3,500,000,000, according to the American Hospital Association survey.

Almost 18,000,000 Americans were hospital patients during the year—one in eight, the survey shows. This was just a million more patients than in the previous year.

Patients didn't stay in general hospitals as long in 1949 as in 1948. The average stay was 8.3 days, or almost a half day less. This was due in part to improvements in medical and hospital care. Because they recovered more quickly, patients can be said to have saved 617,109 days in the hospital, the Association said. At an average $14.33 per day, this would amount to a saving of nearly $8,000,000 to the nation's pocketbook.

More hospital employees were there to care for patients during 1949 too, the survey found. For each 100 patients, short term hospitals (except Federal hospitals) had 169 employees. This was seven more per 100 patients than in 1948. The increase in employees was influenced by the trend to a shorter work week for hospital personnel, and to other continuing improvements in patient care and service.

Total "Book Value" of American Hospitals was almost $7,000,000,000 in September, 1949, a half billion dollars more than in 1948. This amounted to an average investment of $4,800 per bed, the American Hospital Association states. In short term hospitals (except Federal), each bed represents an average investment of $8,255. This is $410 over the 1948 estimated investment. New hospitals and additions built during the year cost many thousands more than $8,255 per bed.

ON THE RECORD—Arizona Blue Shield enrollment passed the 60,000 mark during June—the exact figure at the end of the first half of 1950 being 60,092, a net gain of 8,255 since January 1. The records show that the Blue Shield Plan paid a total of $220,840.75 in subscriber benefits during the first six months of this year, as compared with $302,994.57 during 1949. The 1950 payments covered 4,232 cases. Total of cases in 1949 was 5,425.

Enrollment in Arizona Blue Cross was 115,134 at the end of June, a gain for the six-month period of 5,302. Blue Cross paid 8,131 hospital bills for members in the first half of 1950, with the payments to hospitals for the period totalling $520,871.31. During 1949 Blue Cross paid $912,674.13 to hospitals for members' care, the payments covering 14,355 cases.

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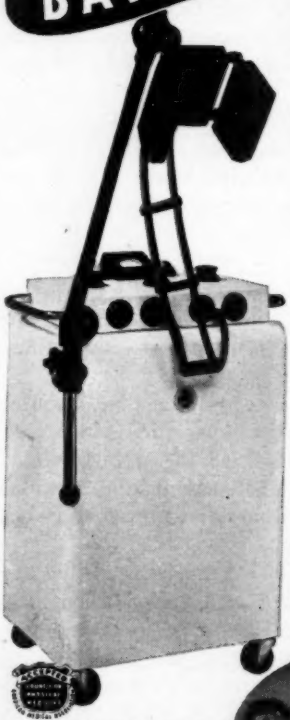

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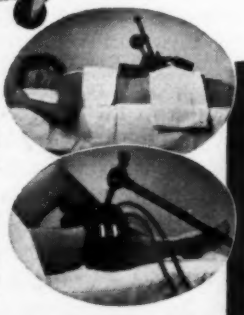

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WOMAN'S AUXILIARY

CONVENTION REPORT

The twenty-seventh Annual Meeting of the Women's Auxiliary to the American Medical Association was held in San Francisco, June 26th to 30th, 1950. The meetings were held in the beautiful Fairmont Hotel, high atop Nob Hill. Seventeen hundred and three members of Auxiliaries from the forty-eight state, District of Columbia and Hawaii were registered. This group represented an organization, which has a membership of 53,606, an increase of 5,000 over last year.

The delegates had little doubt as to the role of Doctors' wives in the year 1950—the pertinent theme of the meeting being, of course voluntary medicine versus compulsory health insurance and how best the Woman's Auxiliary could back the A.M.A. in its fight against government control.

Round Table discussions on TODAY'S HEALTH, Legislation, Public Relations, and Program, at which the National Chairmen served as leaders, were held on Monday. These were most interesting and instructive, as they gave an insight into the many and varied projects which the individual auxiliaries undertake to further their aims—from writing thousands of personal letters to lay organizations to secure endorsements, to forming speakers bureaus, to Florida's super-charged activity in helping to "exterminate" Senator Claude Pepper, (as it was so graphically described by vivacious Mrs. James L. Anderson, President of the Florida State Auxiliary).

Tuesday, June 27th, the Convention was formally opened by the National President, Mrs. David B. Allman. For Mrs. Allman's report, which was given during this meeting, we hope you will read your August BULLETIN. It will give you an idea of how hard and diligently your National Officers work during their year of tenure. Mrs. Allman traveled thousands of miles to visit State Auxiliaries, held countless meetings and conferences and had voluminous correspondence in carrying out the work of the National Auxiliary. She was ably assisted by the President-elect and the officers of the Board and gave them her sincere praise and appreciation.

The reports of the National Officers and Standing Committee Chairmen followed. TODAY'S HEALTH chairman, Mrs. Herbert W. Johnson, produced a most interesting and enlightening chart, which showed that only four State Auxiliaries had reached 100% of their quota, and only six had achieved 50% or over. All the rest of the forty State Auxiliaries fell far below even the 50% mark. She stressed that the Magazine is worthy of more effort and that the doctors should feel a responsibility for ordering it since they asked the Auxiliary to promote it. She also stated that the Auxiliary should do a good job of promotion or admit failure and give the job up. A little more effort on the part of both would soon increase the sale and distribution of a magazine which is an excellent public relations medium. We were delighted to find that Arizona was one of the six states listed as having 72% of its quota, and it is hoped that next year we will go over the top into the 100% group.

The report of the Nominating Committee was read and the following names were on the slate:

Mrs. Harold F. Wahlquist.....Pres.-Elect
Minneapolis, Minn.

Mrs. Leo J. Schaefer.....1st V. P.
Salina, Kansas

Mrs. W. E. Hoffman.....2nd V. P.
Charleston, West Virginia

Mrs. Mason G. Lawson.....3rd V. P.
Little Rock, Ark.

Mrs. John Z. Brown.....4th V. P.
Salt Lake City, Utah

Mrs. George Turner .....Treasurer
El Paso, Texas

Mrs. Charles Shafer .....Constitutional Secretary
Kingston, Pa.

Directors—One Year

Mrs. David B. Allman, Atlantic City, N. J.
Mrs. Scott C. Applewhite, San Antonio, Texas
Mrs. Ralph Eusden, Long Beach, Calif.
Mrs. William W. Potter, Knoxville, Tenn.

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Wednesday, June 28th, the second Session of the General Assembly was held, with Mrs. Allman presiding. After an impressive Memorial service, given by Mrs. Ralph Eusden, the Resolutions committee report was given and approved. These Resolutions included (1) the support of the A.M.A.'s twelve point public health program, and (2) the need to work for the inclusion of instruction on the Constitution of the United States in the curricula of the public schools.

The reports of the State Presidents were next on the agenda. Our State President, Mrs. Benjamin Herzberg, reported for Mrs. Charles E. Starns, the good work done by the Arizona Auxiliaries during the past year, and it received the attention it well deserved. All the state president reports showed clearly that the Woman's Auxiliary is no longer a purely social group, but one which is interested in every type of community endeavor. It is an active, fighting ally of the American Medical Association, from the youngest State Auxiliaries in New Mexico and Maine to the oldest in South Dakota.

The afternoon session saw the election of the National Officers and their installation by Mrs. Rollo K. Packard. As she presented the lovely diamond-studded President's pin to Mrs. Arthur A. Herold, who is, as one of the delegates expressed it, a "Whistler's Mother" type with snap, verve and a southern drawl, we all felt that Mrs. Herold, with her many years of service in the work of the Auxiliary, would carry on the good work done by her predecessors. Mrs. Packard, also, presented the Past President's pin to Mrs. Allman, commending her for the most successful year of great accomplishments which the Auxiliary enjoyed under her leadership. The generous orchid corsages and flowers given to the President and her co-officers made them look very festive.

Mrs. Herold's inaugural address, which will also be in your August BULLETIN, pledged true devotion to the cause and purpose of the American Medical Association and its Auxiliary and an honest endeavor to forward the work already started by her able predecessor. She called for the cooperation and help of the over 50,000 members of the organization, with which she could go ahead to the accomplishment of even greater things.

The Convention was not all work and meetings. A beautiful tea was held on Monday afternoon in honor of the National President and President-elect and State Presidents. A luncheon on Tuesday honored the Past Presidents of the Woman's Auxiliary, at which Dr. Ernest B. Howard, Assistant Secretary of the A.M.A. was the guest speaker. Dr. Howard urged the doctors' wives to push the sale of TODAY'S HEALTH, to help the doctors in their efforts to get the Nursing Associations to back Voluntary Health Insurance and to make resolutions against Compulsory Health Plans, and to work diligently, as citizens, in the forthcoming election in November to register all voters, and urge them to go to the polls and VOTE.

The luncheon on Wednesday, held in the beautiful Peacock Court in the Mark Hopkins Hotel, honored Mrs. Allman and Mrs. Herold. Mrs. Jesse D. Hamer of Phoenix, Arizona, ably presided and introduced the guests of honor, which included Dr. Ernest E. Irons, President of A.M.A.; Dr. Elmer L. Henderson, President-Elect, and others on the National Board of A.M.A.

The Woman's Auxiliary to the California Medical Association is to be congratulated on their hospitality and the wonderful arrangements made for the comfort, entertainment and perfect weather we enjoyed while in their superb City of San Francisco.

Official Delegates to the Convention from Arizona were: Mrs. Harold Kosanke and Mrs. Hiram Cochran of Tucson, and Mrs. Benjamin Herzberg and Mrs. George S. Enfield of Phoenix. Mrs. Royal W. Rudolph, President-elect, attended unofficially, but was present at all the meetings. This report has been compiled from the notes of all the above mentioned, and is duly set down here for your enlightenment and instruction.

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